A Dissertation on

"ROLE OF SURGICAL INTERVENTION IN ACUTE PRESENTATIONS OF TUBERCULAR ABDOMEN IN OUR SETUP"

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY CHENNAI.

with partial fulfilment of the regulations

for the Award of the degree of

M.S. (General Surgery)

Branch – I

MADRAS MEDICAL COLLEGE

CHENNAI-03

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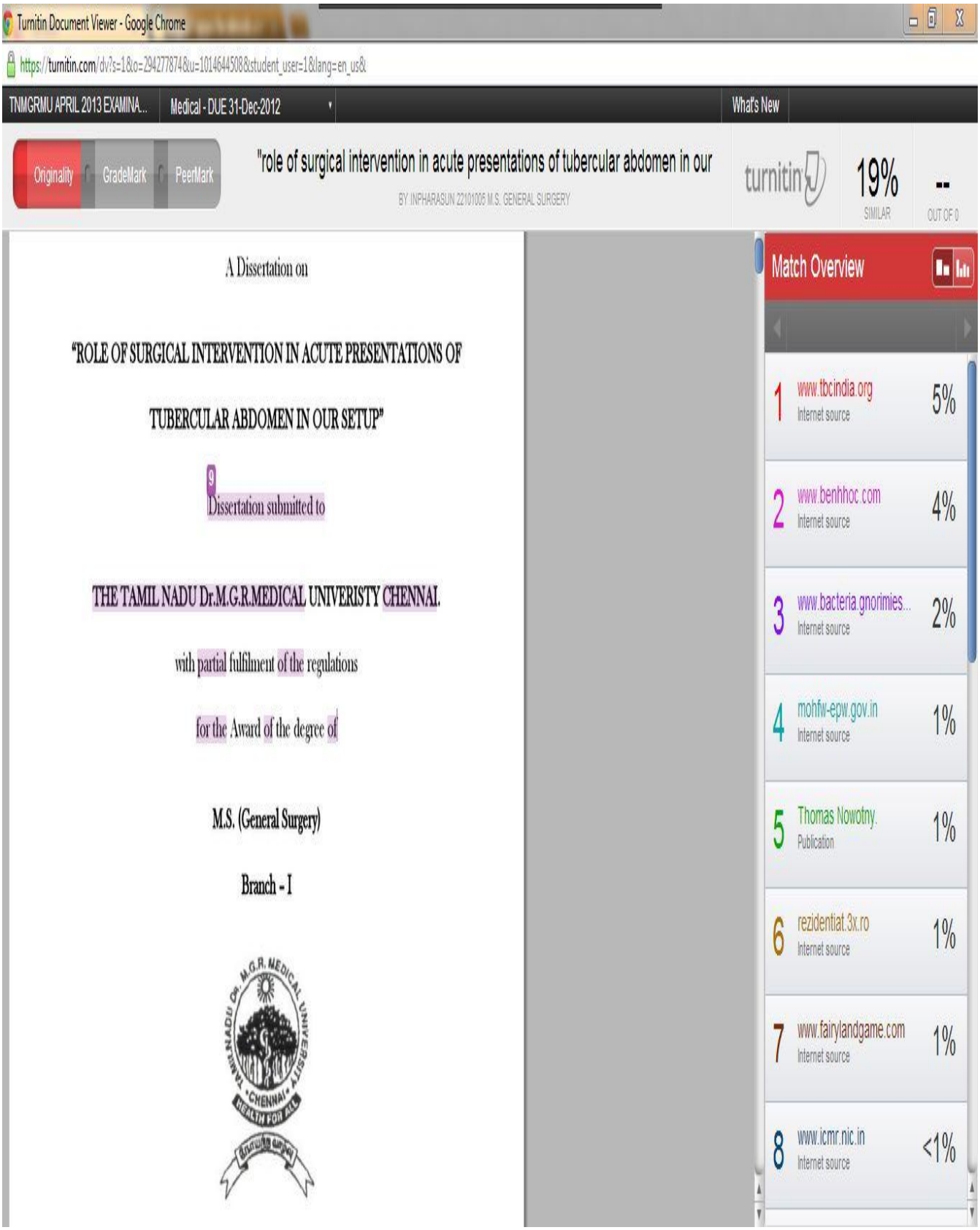
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DECLARATION

I, **Dr. Inpharasun .S.A.,** certainly declare that this dissertation titled **"ROLE OF SURGICAL INTERVENTION IN ACUTE PRESENTATIONS OF TUBERCULAR ABDOMEN IN OUR SETUP""** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice, and are acknowledged.

I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other University board, either in India or abroad. This is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Master of Surgery degree Branch 1 (General Surgery).

Date:

Place: Dr. Inpharasun .S.A.

BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of

Dr. INPHARASUN S.A. on **"ROLE OF SURGICAL INTERVENTION IN ACUTE PRESENTATIONS OF TUBERCULAR ABDOMEN IN OUR SETUP"** during his M.S. (General Surgery) course from May 2010 to April 2013 at the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-03.

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CONTENTS

INTRODUCTION:

Strangely visited people,

All swoln and ulcerous, pitiful to the eye

The mere despair of surgery, he cures,

Hanging a golden stamp about their necks,

Put on with holy prayers: and 'tis spoken,

To the succeeding royalty he leaves

The healing benediction

—Macbeth, Act 4, Scene 3, 171-7

These were the words from Shakespeare's Macbeth which describes the procedure of Royal Touch ceremony performed in medieval ages for treating tuberculosis patients!!!!!!!!!!

Abdomen is one of the common sites of extra pulmonary involvement (6%). An increase in the incidence of pulmonary tuberculosis has led many to predict that there will be a corresponding increase in abdominal tuberculosis. In developing countries abdominal tuberculosis continues to be a common presentation of extra

pulmonary form. However, in developed countries due to increased standards of living the incidence of abdominal tuberculosis has become rare. Since there is an increase in the incidence of Acquired Immuno-deficiency Syndrome (AIDS) recently, the incidence of abdominal tuberculosis and other forms of Extra pulmonary tuberculosis are also expected to rise.

Extra pulmonary form of tuberculosis is difficult to diagnose. Abdominal tuberculosis has bizarre, chronic and insiduous type of presentation and difficult to diagnose. Due to advent of laparoscopy, the diagnosis has been possible without laparotomy. Since there are diverse in clinical presentations, often with equivocal reports of the investigations and wide spread complications, often with prolonged morbidity and mortality, we have analyzed our patients to highlight the various aspects of abdominal tuberculosis in our setup.

The purpose of this study is to find out the commonly encountered causes for emergency laparotomy in known tuberculosis patients presenting as acute abdomen and most frequently performed surgical interventions for the same in our setup.

Rationale behind the study:

Tuberculosis is one of the leading cause of morbidity and mortality all over the world, particularly in a developing country like India. Hence it is not only a health

concern to tackle this deadly & disabling disease but also a major social responsibility.

Among the various clinical presentations of Tuberculosis, most require only medical management. But few, including Abdominal Tuberculosis definitely requires surgical intervention.

Abdominal tuberculosis is like a Pandora's box with a variety of clinical presentations ranging from vague nonspecific abdominal pain to subacute obstruction to characteristic acute abdomen.

If the pathology is diagnosed early , intervention could be done at an earlier stage by ATT thereby avoiding development of acute abdomen or the need for an emergency procedure.

But again, early diagnosis of abdominal tuberculosis is virtually impossible. Patients present only when the disease advances. Most times, they present to the emergency, with obstruction, perforation or abscess.

This is the place, where surgical intervention comes into play. Not only these interventions relieve the patients of their symptoms, but also they provide tissue for diagnosis, thereby prompting the initiation of ATT.

Aims and Objectives:

1. To evaluate the role of surgical interventions in cases of abdominal tuberculosis presenting as emergency
2. To find out the most common surgical presentation of tubercular abdomen as acute abdomen in our setup
3. To find out the most commonly performed surgical intervention in emergency presentations of tubercular abdomen.

Inclusion criteria:

- Known tuberculosis patient presenting as acute abdomen
- Patients presenting as acute abdomen who are found to have tuberculosis after biopsy report

Exclusion criteria:

- Acute abdomen of all other etiologies

REVIEW OF LITERATURE:

History of Tuberculosis:

Since the ancient times Tuberculosis has been mentioned by numerous terms such as Consumption, Phthisis, Scrofula, Pott's disease, the White Plague etc. All these names tell the effect of the disease - Cachexia. Analysis of Mycobacterial Interspersed Repetitive Units inferred that modern day Mycobacterium complex has evolved from a single species nearly 40,000 years ago, the period when Homo sapiens sapiens moved from Africa. And Mycobacterium bovis lineage dispersed approximately 6,000 years ago, the period of animal domestication and early farming. This disease was present in the fossils of Neo-lithic people.

Timeline in discovery of Mycobacterium tuberculosis:

Several breakthroughs happened in 19th century that lead to the discovery of Mycobacterium.

René Laennec- did a lot of studies on Tuberculosis. His works served as beacon for others to work upon.

1869- Jean Antoine Villemin demonstrated that the disease was contagious. He extracted material from human cadavers died of tuberculosis and injected it to lab

rabbits, which got infected with tuberculosis after injection, thereby confirming the contagious nature of the disease.

24 March 1882- Robert Koch discovered Mycobacterium tuberculosis, or Koch's bacillus used a new staining method to stain the sputum of TB patients. Using blood serum he cultured the organism, which he inoculated in laboratory rabbits to reproduce the disease, thereby proving that the tuberculosis bacillus ,was the causative agent of tuberculosis.

1890- Tuberculin, a purified protein derivative was developed from the bacteria as a means of immunization, but it was a failure.

1908-Charles Mantoux discovered that injecting Tuberculin intra dermally was useful investigative tool in diagnosing tuberculosis.

"If the importance of a disease for mankind is measured from the number of fatalities which are due to it, then tuberculosis must be considered much more important than those most feared infectious diseases, plague, cholera, and the like. Statistics have shown that 1/7 of all humans die of tuberculosis"

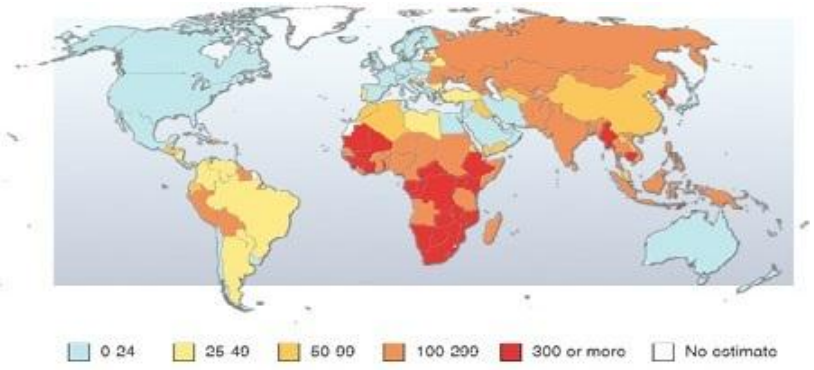
—Die Ätiologie der Tuberculose, Robert Koch (1882)

Epidemiology:

Between 2000 and 2020, it is estimated that, Nearly one billion people will be newly infected with TB, 200 million people will get diseased and at least 35 million lives will be lost all over the world.

India is classified under Group IV countries along with sub-saharan countries, where the incidence and prevalence are very high.

Incidence of tuberculosis in India for the year of 2011 was 181 per 1,00,000 population. The prevalence was 249 per 1,00,000 population. Of these, 19% were Extra pulmonary tuberculosis. 45% of patients with tuberculosis were patient with known HIV status. Abdominal tuberculosis was present in about 6% of the total patients.



world wide incidence per 1,00,000 population

Tamilnadu has very high reporting rates for tuberculosis, all over our country.

In our State, every year about 1.4 lakhs persons develop Tuberculosis, among which 48,000 are sputum positive. Nearly 50 per cent of tuberculosis (TB) patients in Tamil Nadu were found to be having either diabetes or pre diabetes conditions.

Etiologic Agent:

The most common organ that *Mycobacterium tuberculosis* complex infects is the lungs. In up to one-third of cases other organs are involved. With proper treatment, TB is curable by drugs. Untreated, the disease is deadly within 5 years of its onset in half of the cases. Disease is transmitted through droplet nuclei produced by patients with infectious pulmonary TB.

It is an obligate intra cellular bacteria.

Family-Mycobacteriaceae

Order- Actinomycetales.

M. tuberculosis complex,

M. tuberculosis - most common and important agent

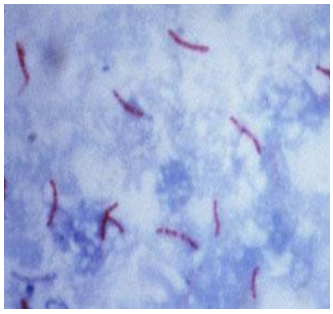
Other organisms of the complex- *M. bovis*, M. *caprae*, *M. africanum*,

M. microti , *M. pinnipedii* , and *M. canetti* .

M. tuberculosis, nonsporing, aerobic acid fast bacilli. Mycolic acid(long chained,cross linked fatty acids) are present in the bacterial cell wall.They are linked to the arabinogalactan and peptidoglycan molecules, present below. not only this produces acid fastness of the bacilli, but also they are responsible for very low permeability of the cell wall, & reduced susceptibility to most antibiotics.

The molecule lipoarabinomannan is responsible for the bacteria-macrophage interaction and it aides in survival of the bacteria inside macrophage.

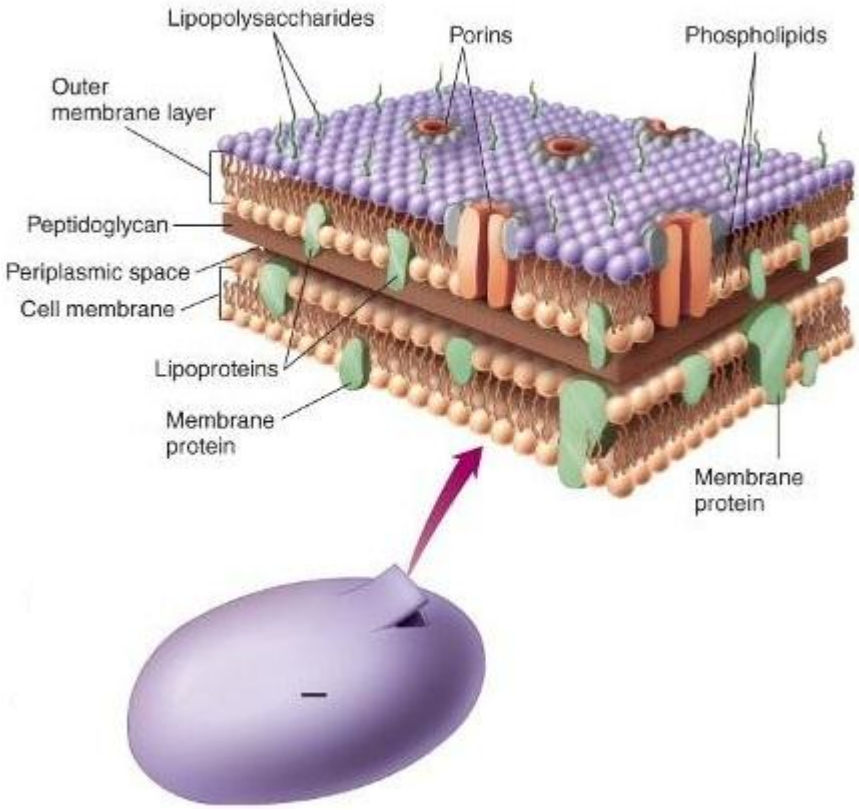
Acid-fast bacillus smear



These Bacilli can be cultured ex-vivo using Löwenstein–Jensen medium & BACTEC medium.

Löwenstein–Jensen medium

Bacterial cell wall structure:



Pathogenesis:

From Exposure to Infection:

Exposure leading to Infection- it depends mainly on the exogenous factors,which are....

- Crowding,
- Poor ventilation,
- Over population.

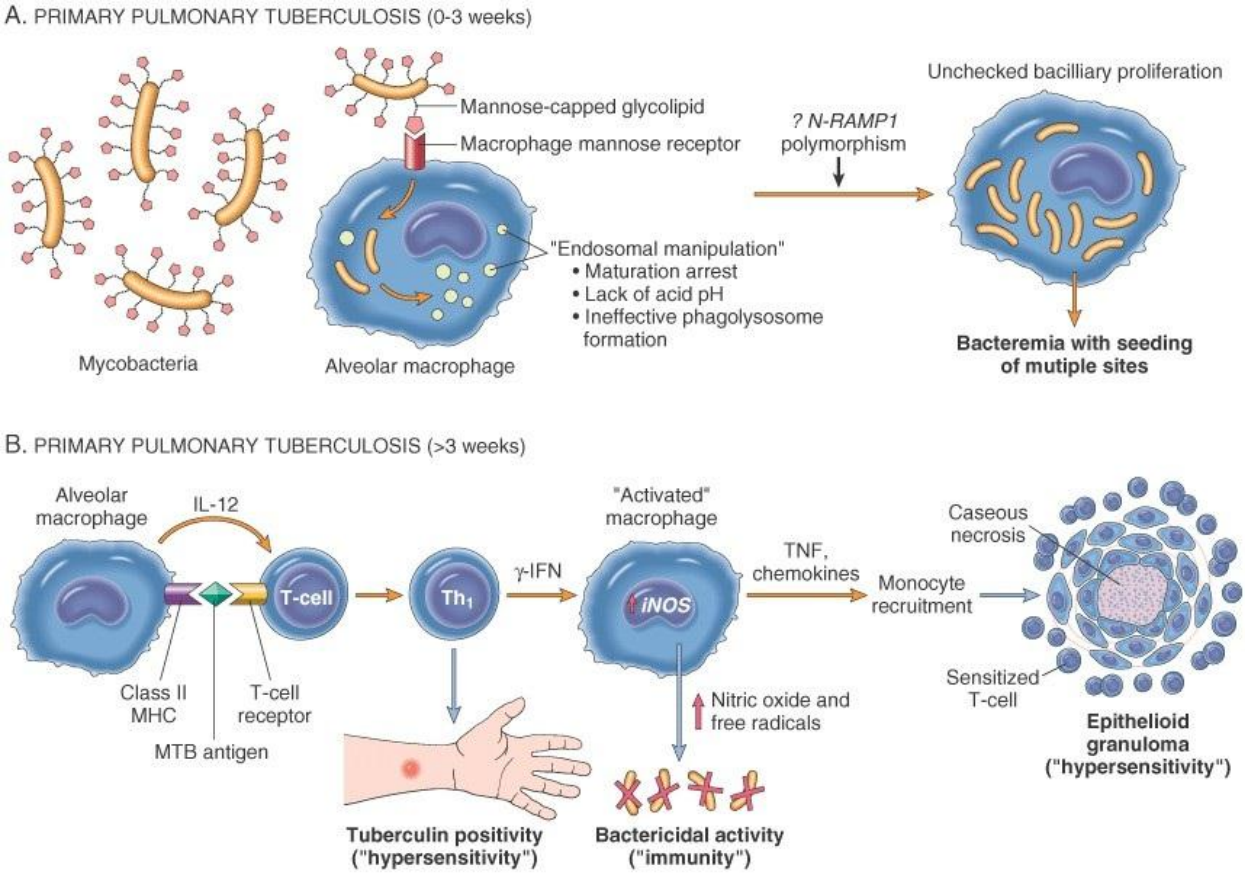
These exogenous factors determine the risk of acquiring *M. tuberculosis* infection.

Organism is transmitted from a person to person by droplet nuclei. Each cough may have as many as 3000 infectious nuclei. Apart from that it can be transmitted through the skin contact & placenta(of course both have minimal role only)

PTB patients, who are sputum positive are the ones who transmit the infection mostly. Majority of the patients spreading the disease have cavitary lung disease but few have laryngeal TB.

Sputum smear–negative/culture-positive TB patients are less infectious.

Culture-negative pulmonary TB and extrapulmonary TB wont infect any. Persons with both HIV infection and TB have less cavitations, and they are essentially less infectious than those without HIV infection.


A. PRIMARY PULMONARY TUBERCULOSIS (0-3 weeks)
Mycobacteria
Mannose-capped glycolipid
Macrophage mannose receptor
Alveolar macrophage
"Endosomal manipulation"
• Maturation arrest
• Lack of acid pH
• Ineffective phagolysosome formation
? N-RAMP1 polymorphism
Unchecked bacilliary proliferation
Bacteremia with seeding of mutiple sites
B. PRIMARY PULMONARY TUBERCULOSIS (>3 weeks)
Alveolar macrophage
IL-12
T-cell
Class II MHC
T-cell receptor
MTB antigen
Th1
γ-IFN
"Activated" macrophage
iNOS
TNF, chemokines
Monocyte recruitment
Caseous necrosis
Sensitized T-cell
Epithelioid granuloma ("hypersensitivity")
Nitric oxide and free radicals
Tuberculin positivity ("hypersensitivity")
Bactericidal activity ("immunity")

From Infection to Disease:

Infection leading to disease- it depends on endogenous factors, which are....

i. Innate immunity
ii. Cell-mediated immunity (CMI).

Primary TB – clinical disease that occurs after initial infection (common among young children and immune compromised).

Primary TB is severe and disseminated, but generally it has only low-level transmissibility.

When infected in later years of life, mature immune system of the older age group will control the evolution into primary PTB.

Secondary (or *postprimary*) ***TB*** is often more infectious than primary disease, because of cavitation. This is most commonly due to reactivation of dormant bacilli.

Of the patients infected primarily up to 10% will develop active TB in their lifetime, 50% during the first year after infection.

HIV-infected patients have higher infection rate.

In high prevalence regions, Secondary TB is due to reinfection of a previous individual.

Majority of the PTB patients in our country are secondary TB patients.

Determinants of development of disease after infection include

- Age
- Immune status
- HIV co-infection

The disease can be cured in majority of patients.

With effective, timely, and proper drugs, patients have a very high chance of cure.

Infection and Macrophage Invasion:

Host and Bacterial interaction starts with inhalation of droplet nuclei.Droplet nuclei are produced from the cough of the infected patients, which contain the bacteria.

Upper airways trap and expel most of the bacteria (almost 90%)

Only <10% reach the alveoli.

Inside alveoli, alveolar macrophages phagocytize the bacilli.

Mycobacteria -macrophage interaction starts with the binding of the bacterial cell wall with macrophage cell-surface molecules, which are,

- complement receptors,
- the mannose receptor,
- the immunoglobulin GFc receptor,
- type A scavenger receptors

Complement pathway gets activated releasing C3 activation products such as C3b, which opsonize the bacilli making them tasty for the phagocytes to phagocytosize leading to phagosome formation. Inside the phagosome,

M. tuberculosis reduces acidification by inhibiting the accumulation of vesicular proton-adenosine triphosphatase.

Lipoarabinomannan inhibits the increase of calcium inside the host cell and affects phagosome-lysosome fusion, thereby leading to the survival of the bactera inside the host cell.

Once bacilli survives inside the cell, it will start multiplying eventually rupturing the phagocyte and releases its contents. These contents include potent cytokines which leads to recruitment of other uninfected phagocytic cells.

These recruited cells continue the infection cycle by ingesting dying macrophages and their bacillary content, and in turn becoming infected themselves thereby expanding the infection.

Virulence of Bacilli:

***katG* gene-** gene responsible for producing catalase/peroxidase enzyme. This enzyme protects from oxidative stress& converts isoniazid to active form.

Two key small protein antigens

(ESAT-6) --early secretory antigen-6

(CFP-10) -- culture filtrate protein-10

facilitate the egress of the bacterium from host cell.

These proteins are absent in the vaccine strain *M. bovis* (BCG) has been shown to be a key attenuating mutation.

Isocitrate lyase gene *icl1* --encodes for an enzyme important in the glyoxylate shunt

This gene is required for long-term persistence of *M. tuberculosis* infection in mice with chronic TB.

carD -- gene that encodes for recently identified mycobacterial protein CarD

This protein is essential for the control of rRNA transcription required for replication and persistence in the host cell.

Loss of this gene exposes mycobacteria to oxidative stress, starvation, DNA damage, and ultimately sensitivity to killing by a variety of host mutagens and defensive mechanisms.

Host Response:

In the first phase of infection, *M. tuberculosis* undergoes a period of extensive growth within naïve unactivated macrophages, and additional naïve macrophages leading to the formation of early granuloma.

This is before the activation of cell mediated immunity.

M. tuberculosis has specific virulence mechanisms that subverts host cellular signaling and elicits an early proinflammatory response that promotes granuloma expansion and bacterial growth during early phase.

Mycobacterial protein ESAT-6 induces secretion of matrix metalloproteinase 9 (MMP9) by nearby epithelial cells that are in contact with infected macrophages. MMP9 in turn stimulates recruitment of naïve macrophages, thus inducing granuloma maturation and bacterial growth. Disruption of MMP9 function results in reduced bacterial growth.

Cytokines and bacterial cell products that are produced repeatedly from frequent lysis of cells, cause the migration of naive macrophages.

These macrophages too will get infected and subsequently infect the dendritic cells, which they recruit.

These cells enter the nearby draining lymph nodes and present the antigens from the bacteria to T lymphocytes.

This is the time of development of Cell Mediated Immunity and humoral immunity.

These initial stages of infection are usually asymptomatic.

Host responses are of 2 types (which usually takes about 2-4weeks to start):

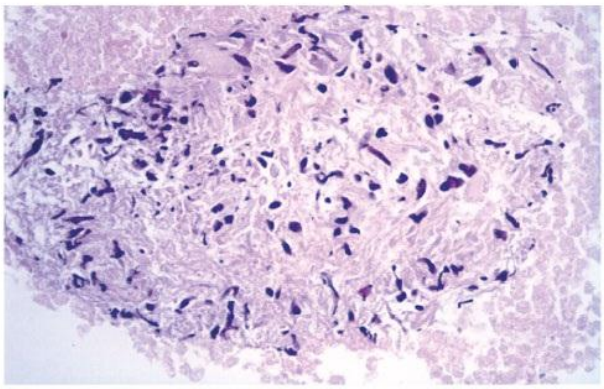
1) Macrophage-activating CMI response&
2) Tissue-damaging response

The *macrophage-activating response* - T cell–mediated

- activates macrophages that kill and digest the bacilli.
- limits mycobacterial growth within macrophages
- macrophages stimulate T lymphocytes to release lymphokines
- T lymphocytes mediate futhur inflammatory reaction
- causes central *caseous necrosis* - lesion where activated macrophages occupy the lesion's middle thereby effectively neutralizing tubercle bacilli with minimal host tissue damage
- Even inside the healed lesions, viable organism may remain dormant within macrophages for many years.
- These lesions may undergo eventual calcification.

The *tissue-damaging response* - delayed-type hypersensitivity (DTH) reaction

- destroys unactivated macrophages that contain bacilli
- this is only in few percentage of patients where CMI is weak
- produces early solid necrosis at the site of lesion
- this will lead to tissue destruction
- also there will be erosion of blood vessels, bronchioles and nearby lymph nodal spread
- this might lead to the disseminated disease
- in patients with immature immune system such as young children and in immuno compromised, the disease will be severe and often fatal



Histological examination shows coagulation necrosis and epithelioid cells (HE stain, ×100).

Both responses inhibit bacterial growth, but the balance between the two responses determine what sort of TB the patient is going to develop.

Specific immunity develops at a later stage where large number of activated macrophages accumulated near the primary lesion leading to the formation of granulomas.

It is possible that an immune response capable of eradicating early infection may sometimes occur due to disabling mutations in mycobacterial genomes rendering their replication ineffective.

Macrophages and Monocytes:

Cell Mediated Immunity - provides partial protection against *M. tuberculosis*

- T lymphocytes, Macrophages and Monocytes are responsible
- Macrophages ingest bacilli and try to destroy them through a nitric oxide mediated mechanism
- Also they release cytokines that recruit other inflammatory cells.
- These include Interferon, Tumor Necrosis Factor and Interleukin-1

Humoral immunity - provides very little role in protection.

T Lymphocytes:

Alveolar macrophages, monocytes, and dendritic cells, all these cell process the bacilli they ingest and present the antigens to T lymphocytes. Both CD4+ and CD8+ T cells.

The activation and proliferation of CD4+ T lymphocytes are important, because their role is crucial to host's defense against *M. tuberculosis*. Defective CD4+ T-lymphocytes fail to contain the proliferation of tubercle bacilli.

Mycobacterial Lipids and Proteins:

Blood borne Dendritic cells recognise the lipids present on the bacillary cell wall through Toll like receptor(TLR) and lead to activation of innate immune system.

Among the various antigens present in the bacilli, secreted antigens elicit T lymphocyte response.

Tuberculosis:

- Pulmonary
- Extra Pulmonary
- Both

80% were pulmonary in non HIV patients.

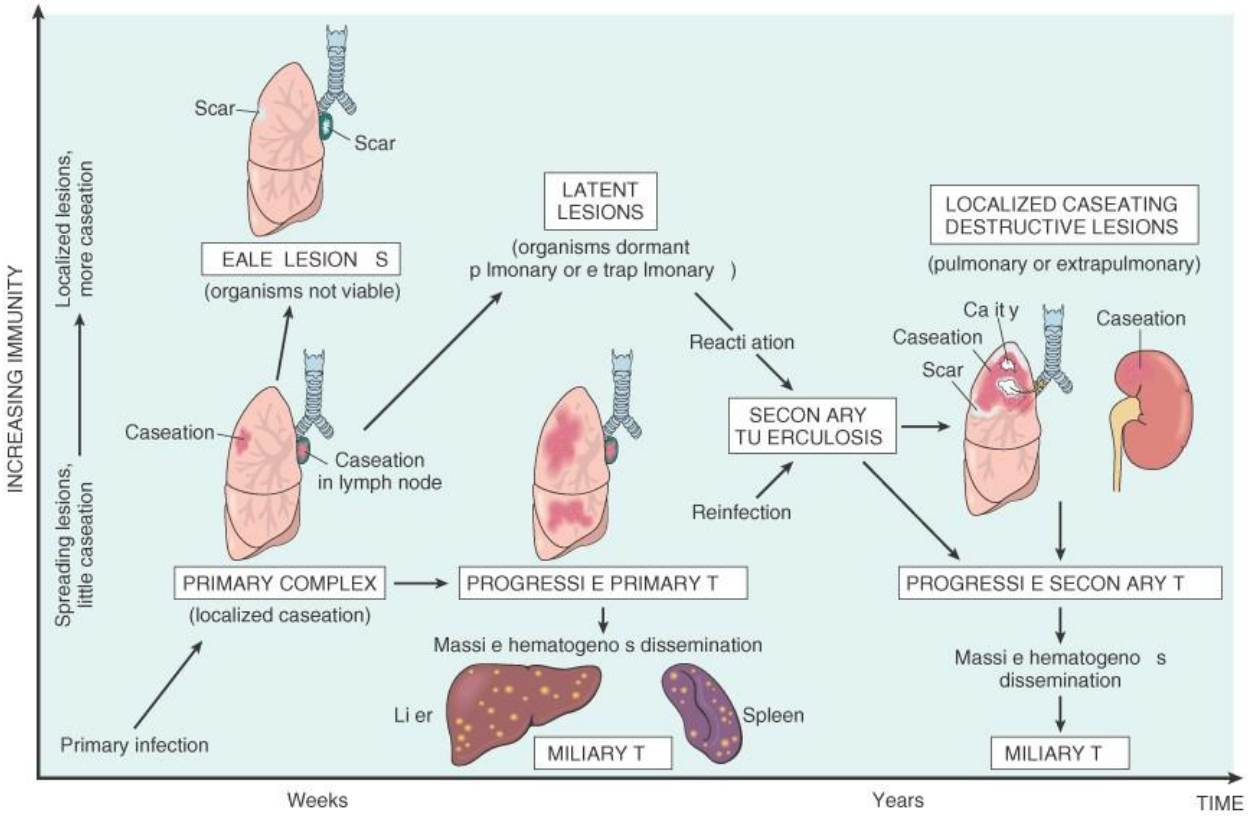
But 60% of HIV patients with TB have both pulmonary and extrapulmonary TB or extrapulmonary TB alone.

PTB:

Pulmonary TB can be

- primary or (disease following first infection)
- postprimary (adult-type, secondary).(disease following either reactivation or re infection)

SYSTEMIC SPREAD OF TB FROM LUNGS



Primary PTB:

Primary PTB -occurs after the initial infection, often seen in children. Affects the middle and lower lung zones and patient may have pleuritic chest pain but most patients are asymptomatic. Initial infection (the Ghon focus) is a peripheral lesion, accompanied by transient hilar or paratracheal lymphadenopathy. Pleural reaction may also occur if the focus is sub pleural. The Ghon focus+ regional lymphadenopathy, is called *Ghon complex*.

If the Cell Mediated Immunity is immature or impaired primary PTB progresses rapidly (*progressive primary TB*).

Postprimary (Adult-Type) PTB:

Reactivation or *secondary TB postprimary TB* - is *adult-type TB*.

Post primary can either due to re activation or re infection.

The lung disease varies, from small infiltrates to extensive cavitary disease.

Extrapulmonary TB:

Extra pulmonary tuberculosis involves the following sites with decreasing order of frequency.

- Lymph nodes,
- Pleura,
- Genitourinary tract,
- Bones and joints,
- Meninges,
- Gastro intestinal,
- Pericardium.... etc

Any organ systems can be affected.

Extrapulmonary TB is seen more common today than earlier.

Lymph Node TB (Tuberculous Lymphadenitis):

Lymph nodes are most common presentation of extrapulmonary TB in both HIV & non HIV patients.

Patients present with painless swelling of the lymph nodes, commonly involving posterior cervical and supraclavicular sites (*scrofula*).

Pleural TB:

Contributes to 20% of extrapulmonary cases. Presents as

- Isolated pleural effusion &
- Pleurisy.

Patient may have symptoms such as fever, pleuritic chest pain, and dyspnea. Pleural fluid analysis essential for the diagnosis. The fluid is straw colored, hemorrhagic, exudative.

Tuberculous empyema is a less common complication of pulmonary TB. Drainage is usually required along with ATT. It may lead to severe pleural fibrosis and restrictive lung disease. Removal of the thickened visceral pleura (decortication) is occasionally necessary to improve lung function.

TB of the Upper Airways:

Secondary to advanced pulmonary TB with cavitary lesions. It involves the larynx, pharynx, and epiglottis. Hoarseness of voice, change of voice, difficulty in swallowing, chronic productive cough are the symptoms. Often it mimics carcinoma of larynx.

Genitourinary TB:

Genitourinary TB - 10–15% of all extrapulmonary cases.

Any part of the genito urinary tract can be involved. Dysuria, hematuria, and flank or abdominal pain are common symptoms. Urinalysis is abnormal in 90% of cases, with pyuria (pyuria in acidic urine without any growth) and hemeturia.

Genital TB is present commonly in females than males.

In females it affects the fallopian tubes& endometrium.

It might cause Infertility, pelvic pain, and menstrual abnormalities.

In males genital TB preferentially affects the epididymis, which may drain externally through a fistula.

It can also cause orchitis and prostatitis.

Genitourinary TB responds well to chemotherapy.

MRI kidneys showing tuberculous kidneys

Skeletal TB:

TB of the bones and joints are involved in 10% of extra pulmonary TB.

Disease is due to reactivation of previous foci or spread from adjacent paravertebral lymph nodes.

Hip and knee joints are most commonly affected.

Spinal TB involves two or more adjacent vertebral bodies.

A paravertebral "cold" abscess may also occur. A catastrophic complication of Pott's disease is paraplegia. Paraparesis due to a large abscess is a medical emergency and requires rapid drainage. Skeletal TB responds to chemotherapy, but severe cases may require surgery.

Pericardial TB:

Pericardium is affected in following ways

- direct extension from adjacent mediastinal/hilar lymph nodes or
- hematogenous spread.

The effusion is exudative in nature, with a high count of lymphocytes and monocytes. Hemorrhagic effusion is common.

TB myocarditis is due to direct extension from the pericardium. It is very rare and very fatal.

Miliary or Disseminated TB:

Miliary TB is due to hematogenous dissemination of Mycobacterium tuberculosis.

- In children -primary infection,
- In adults -reinfection or reactivation.

The lesions are yellowish granulomas which resemble millet seeds and are 1–2 mm in diameter.

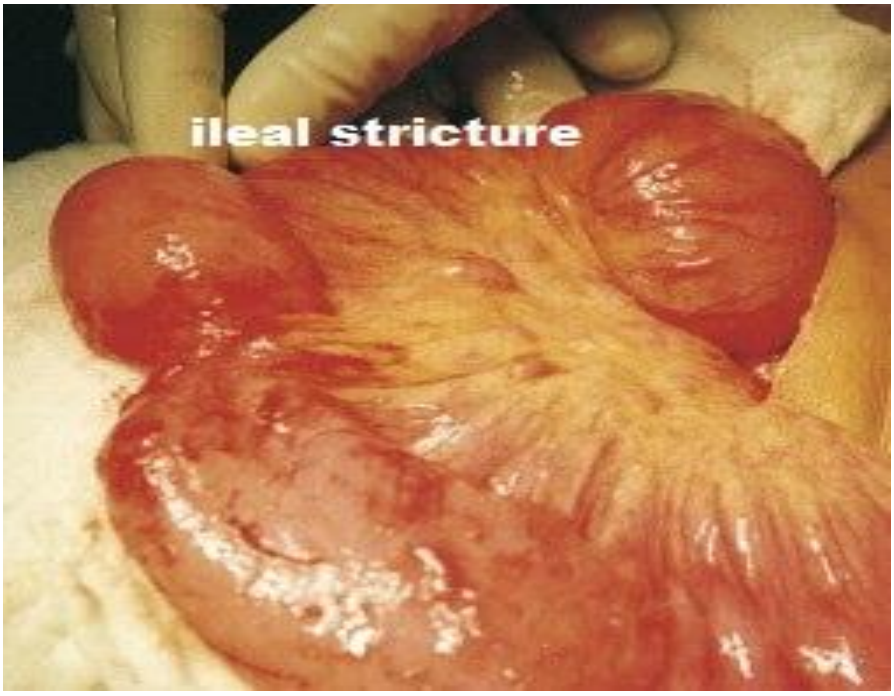
Fever, night sweats, anorexia, weakness, and weight loss are presenting symptoms in the majority of cases. Physical findings include hepatomegaly, splenomegaly, and lymphadenopathy.

Gastrointestinal TB:

Any part of the gastrointestinal tract can be affected by tuberculosis.

GIT is the sixth most frequent site of extra pulmonary involvement.

Spread is due to swallowing of sputum with direct seeding, hematogenous spread, or ingestion of milk from cows affected by bovine TB.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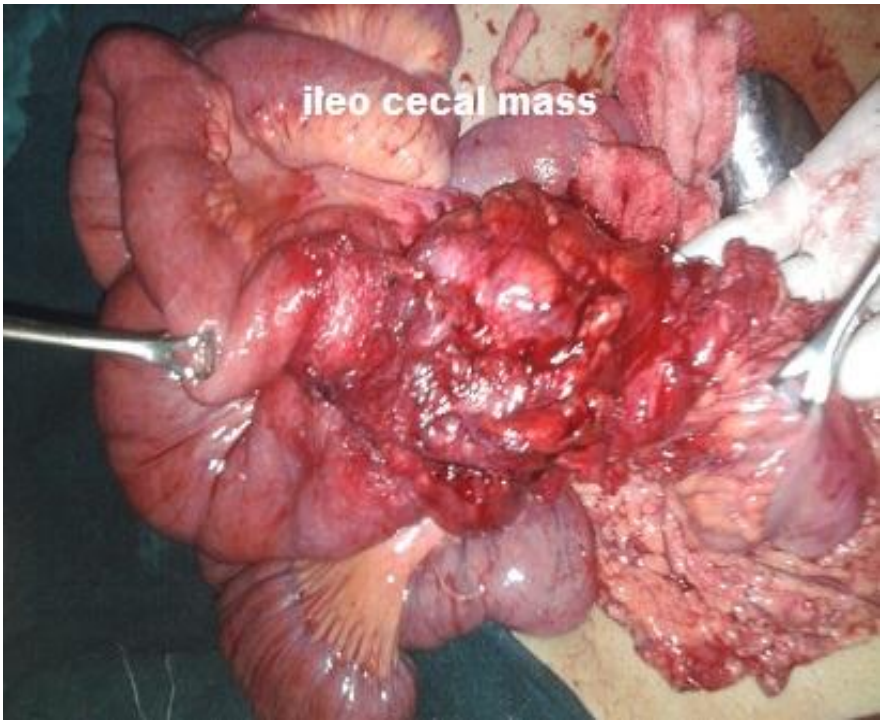
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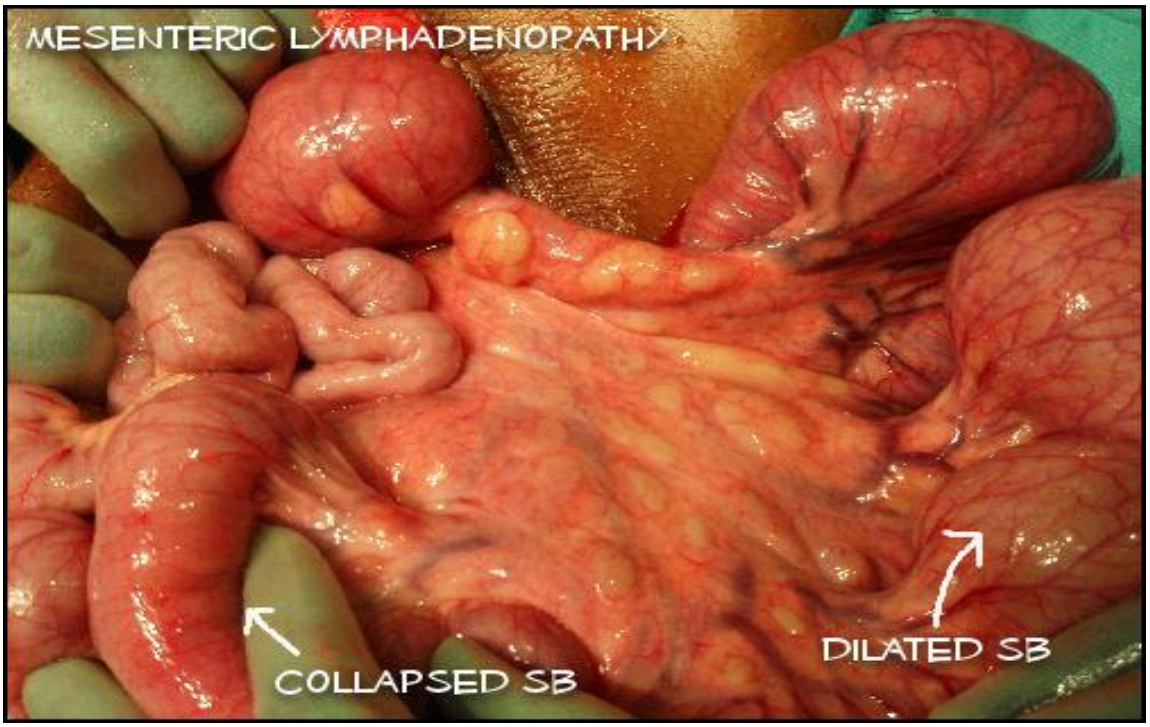


Various presentations of abdominal tuberculosis are

1. Intestinal

    1) Ileo- Caecal tuberculosis

        a) Ulcerative type

        b) Ulcero- Hyperplastic

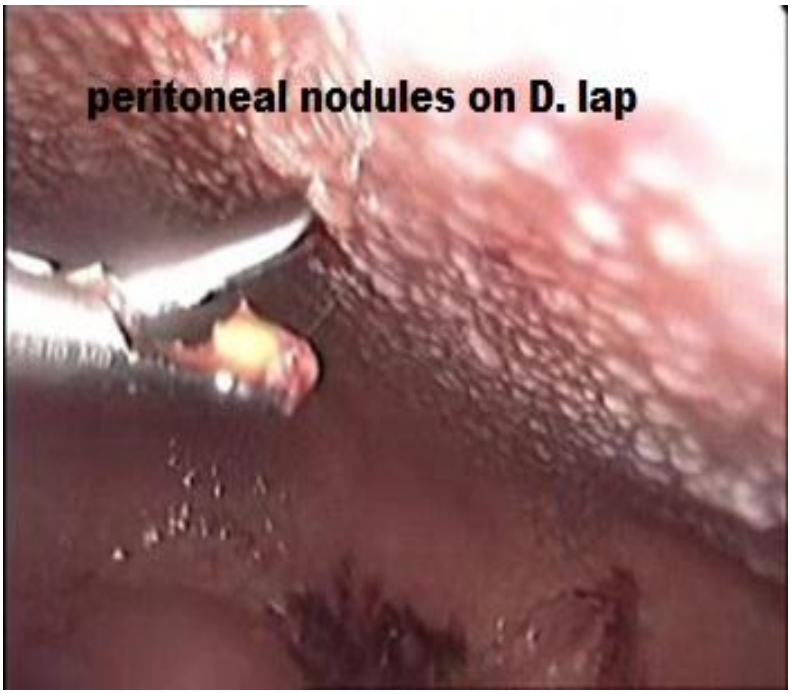
        c) Hyperplastic

    2) Ileal tuberculosis - strictural disease

2. Peritoneal tuberculosis

1) Acute


peritoneal tuberculosis


peritoneal nodules on D. lap

2) Chronic

a) Ascitic type

b) Encysted (loculated) type

c) Plastic (Fibrous/Adhesive) type

d) Purulent type

3. Mesentric tuberculosis

4. Ano-Recto-Sigmoidal tuberculosis

5. Visceral Miliary Tuberculosis

6. Omental tuberculosis

7. Others

Retro Peritoneal

Gastro- Duodenal

The disease is characterized by the presence of transverse ulcers of the mucosa but the inflammatory process maybe transmural.

Stricture of the intestine, particularly that of ileum is common.

Terminal ileum and the cecum are most commonly involved sites though it can affect anywhere in GIT. Abdominal pain, swelling, obstruction, and a palpable mass in the abdomen are common findings at presentation.

Tuberculous peritonitis is due to the

- Direct spread of tubercle bacilli from lymph nodes and viscera
- Hematogenous seeding.
- Nonspecific abdominal pain, fever, and ascites.

1) Mesenteric lymph nodes will be enlarged, matted and may often undergo caeseous necrosis, thereby leading to tubercular abscess.
2) Omental thickening and peritoneal tubercles are other presentations.

In tuberculous peritonitis, paracentesis reveals an exudative fluid with a high protein content and leukocytosis that is usually lymphocytic (although neutrophils occasionally predominate). The yield of direct smear and culture is relatively low; culture of a large volume of ascitic fluid can increase the yield, but peritoneal biopsy (with a specimen best obtained by laparoscopy) is often needed to establish the diagnosis.

Ascitic fluid analysis have the following characteristics:

a. Appearence: straw coloured fluid,
b. Protein content: High,
c. Serum ascitis albumin gradient < 1.1 g/dl,
d. Cytology: predominantly lymphocytic cells,

e. Adenosine deaminase > 36 U/l.

In cases of equivocal diagnosis Diagnostic Laparoscopy is a very useful.

In GIT the ileocaecal region is most commonly affected by tuberculosis.

Patients present with a palpable mass in the right iliac fossa with/without obstruction/perforation cachexia.

Other rare presentations are

i. Oesophageal ulcer

ii. Gastroduodenal tuberculosis,

iii. Colonic tuberculosis,

iv. Rectal stricture and multiple perianal fistulae due to rectal and anal involvement.

< 25 per cent of patients have lung lesions in chest X rays.

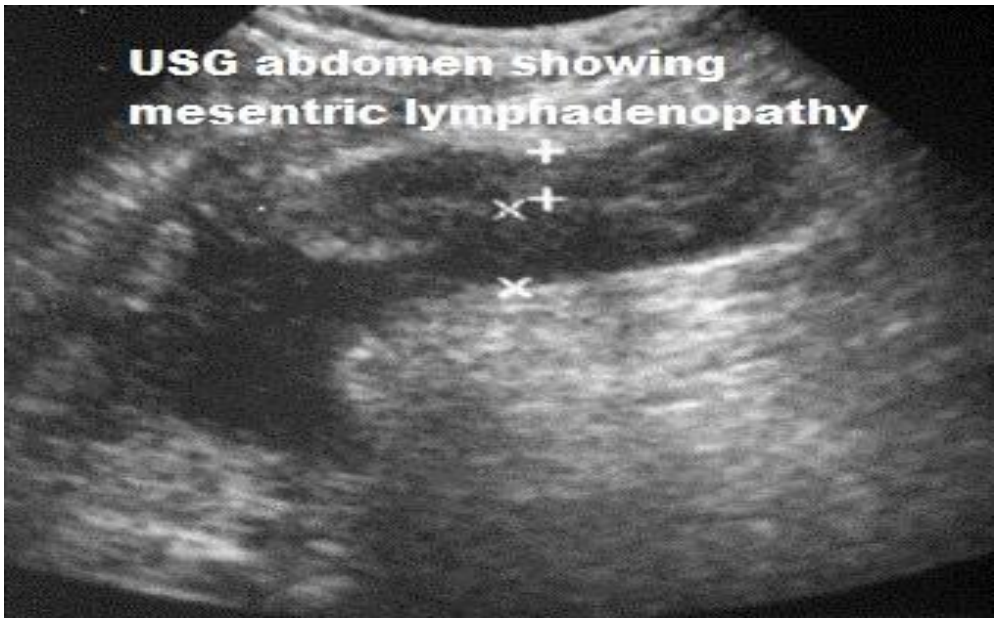
Fever, weight loss, anorexia, and night sweats are also common.

Abdominal TB often mimics Crohn's disease.

CECT IMAGE OF ILEO CECAL TUBERCULOSIS

X RAY ABDOMEN- INTESTINAL OBSTRUCTION

USG ABDOMEN



Investigations:

i. ultrasonography,

ii. small bowel barium meal,

iii. barium enema,

iv. computed tomographic scan and

v. colonoscopy.

In acute presentations of abdominal tuberculosis patients usually present with fever and abdominal pain, vomiting, consipation/ obstipation with abdominal distention suggesting intestinal obstruction.

Management:

Surgical interventions performed were,

- Stricturoplasty
- Resection Anastomosis of Ileal stricture with/without proximal diversion ileostomy
- Limited Resection of Ileum, Caecum & Ascending Colon with Ileo-Ascending anastomosis with/without proximal diversion ileostomy
- Right Hemicolectomy with/without proximal diversion ileostomy
- Extended Right Hemicolectomy
- Mesentric Lymph Node biopsy
- Diversion ileostomy.

Stricturoplasty:

The wall of the strictured bowel is incised longitudinally. Reconstruction is performed by closing the defect.This is usually done for very small strictures.

Resection and anastomosis:

The stricturous portion along with the proximal and distal portion are resected for a short length with end to end primary anastomosis.

Covering proximal diversion ileostomy can be done.

Limited Resection:

For Ileo ceacal tuberculosis, the diseased portion, along with small distance of ileum and ascending colon is resected and end to side or end to end primary anastomosis is done.

Diversion Ileostomy:

When the peritoneal cavity is grossly contaminated or when there is difficulty in performing a definitive procedure, diversion procedures can be performed to relieve obstruction.

Role of Diagnostic Laparoscopy:

Wth the increased availability of laparascopy, diagnostic laparascopy has become a principle modality of investigation for many non specific abdominal complaints, after all other non invasive tests do not show any conclusive evidence.

The advantage of this minimally invasive technique is that it avoids unnecessary laparotomies and at the same time helps to diagnose abdominal tuberculosis at an early latent stage.

Moreover it could be used for intervention including taking biopsy for tissue proof in doubtful cases.

ATT REGIMEN:

RNTCP has revised the previous regimen thereby discontinuing Category III.

Revised categories

Treatment groups	Type of patient	Regimen	
		Intensive Phase (IP)	Continuation Phase (CP)
New (Cat I)	New Sputum smear-positive New Sputum smear-negative New Extra-pulmonary New Others	$2H_3R_3Z_3E_3$	$4H_3R_3$
Previously Treated (Cat II)	Smear-positive relapse Smear-positive failure Smear-positive treatment after default Others	$2H_3R_3Z_3E_3S_3$/ $1H_3R_3Z_3E_3$	$5H_3R_3E_3$

H- ISONIAZID Z- PYRAZINAMIDE S- STREPTOMYCIN

R- RIFAMPICIN E- ETHAMBUTOL

Just providing anti-TB medication is not sufficient to ensure that patients are cured. The DOTS strategy ensures that infectious TB patients are diagnosed and treated effectively till cure, by ensuring availability of the full course of drugs and a system for monitoring patient compliance to the treatment.

Directly Observed Treatment, Short-course (DOTS)

The DOTS strategy was implemented under the Revised National Tuberculosis Control Programme (RNTCP) in India, for comprehensive TB control.

Every day under the RNTCP, more than 15,000 suspects are being examined for TB.

The diagnosis of these patients and the follow-up of patients on treatment is achieved through the examination of more than 50,000 laboratory specimens. As a result, each day about 3,500 patients are started on treatment, stopping the spread of TB in the community.

As a result of rapid expansion in diagnostic facilities, the proportion of sputum-positive cases confirmed in the laboratory are double that of the previous programme and is on par with international standards. Despite the rapid expansion, overall performance remains good and in many areas is excellent. Treatment success rates have tripled from 25% in the earlier programme to 86%in RNTCP.

TB is the most common opportunistic infection in people living with HIV virus. As the HIV breaks down the immune system, HIV- infected people are at greatly increased risk of TB.

Without HIV, the lifetime risk of developing TB in TB-infected people is 10%, compared to at least 50% in HIV co-infected.

HIV is also the most powerful risk factor for progression from TB infection to TB disease. TB in turn accelerates the progression of HIV to AIDS and shortens the survival of patients with HIV infection.

With large numbers of HIV-positive individuals in India, it is likely that HIV may worsen the TB epidemic in the absence of a robust TB control programme.

However, even among HIV-infected people, TB can be cured. Directly Observed Treatment, Short-course (DOTS) is as effective among HIV- infected TB patients as among those who are HIV negative.

Multi-drug-Resistant Tuberculosis (MDRTB) :

MDR TB refers to strains of the bacterium which are proven in a laboratory to be resistant to the two most active anti-TB drugs, isoniazid and rifampicin. Treatment of MDR TB is extremely expensive, toxic, arduous, and often unsuccessful.

DOTS has been proven to prevent the emergence of MDR TB, and also to reverse the incidence of MDR TB where it has emerged. MDR TB is a tragedy for individual patients and a symptom of poor TB management. The best way to confront this challenge is to improve TB treatment and implement DOTS.

Introduction of DOTS-Plus:

The first WHO endorsed DOTS-Plus programmes began in 2000. DOTS-Plus pilot projects have demonstrated the feasibility and effectiveness of MDR-TB treatment.

DOTS-Plus projects have multiplied rapidly.

Multi-drug resistant Tuberculosis (MDR-TB) is defined as resistance of Mycobacterium tuberculosis to Rifampicin and Isoniazid, two of the most effective anti-tubercular drugs for treatment of Tuberculosis.

MDR-TB levels are about 3% in new cases and 12%-17% in re-treatment cases.

Although the level of MDR-TB in the community is low, in relation to percentages and proportions it translates into large absolute numbers.

To address this issue the Revised National Tuberculosis Control Programme (RNTCP) has initiated the DOTS Plus strategy for appropriate management of MDR TB patients and to prevent the propagation and dissemination of MDR-TB.

Category IV regimen:

Intensive phase:

Kanamycin, Ofloxacin, Ethionamide, Pyrazinamide, Ethambutol, Cycloserine (6-9 months)

Continuation phase.

Ofloxacin, Ethionamide, Ethambutol, Cycloserine (18 months)

Materials and Method:

This study was conducted in RGGGH & Madras medical college, Department of general surgery during the period of August 2012 – December 2012. Informed written consent was obtained from all the patients. All the base line investigations comprising haematological and biochemical parameters had been done. about 60 patients who were admitted to the emergency department between may 2010 and june 2012 with acute abdominal complaints were taken for the study. The diagnosis of abdominal tuberculosis were made by clinical findings and investigational reports including histo pathological report of the specimen. All the patients underwent emergency X ray chest PA view, X ray abdomen erect view, USG abdomen. Some selected patients were subjected to CECT abdomen. The surgical procedures done were, laparotomy and mesentric node biopsy/ omental/ peritoneal biopsy, limited resection of ileum, cecum and ascending colon with ileo colic anastomosis with & without proximal diversion, resection and anastomosis of ileum with or without proximal diversion, stricturoplasty, adhesiolysis with peritoneal biopsy. With consent patients were subjected to HIV 1&2 ELISA. All the patients underwent sputum AFB post operatively to look for pulmonary foci and patients were subjected to Cat 1 or Cat 2 ATT as per pulmonary involvement. Patients were followed up post operatively till the completion of ATT.

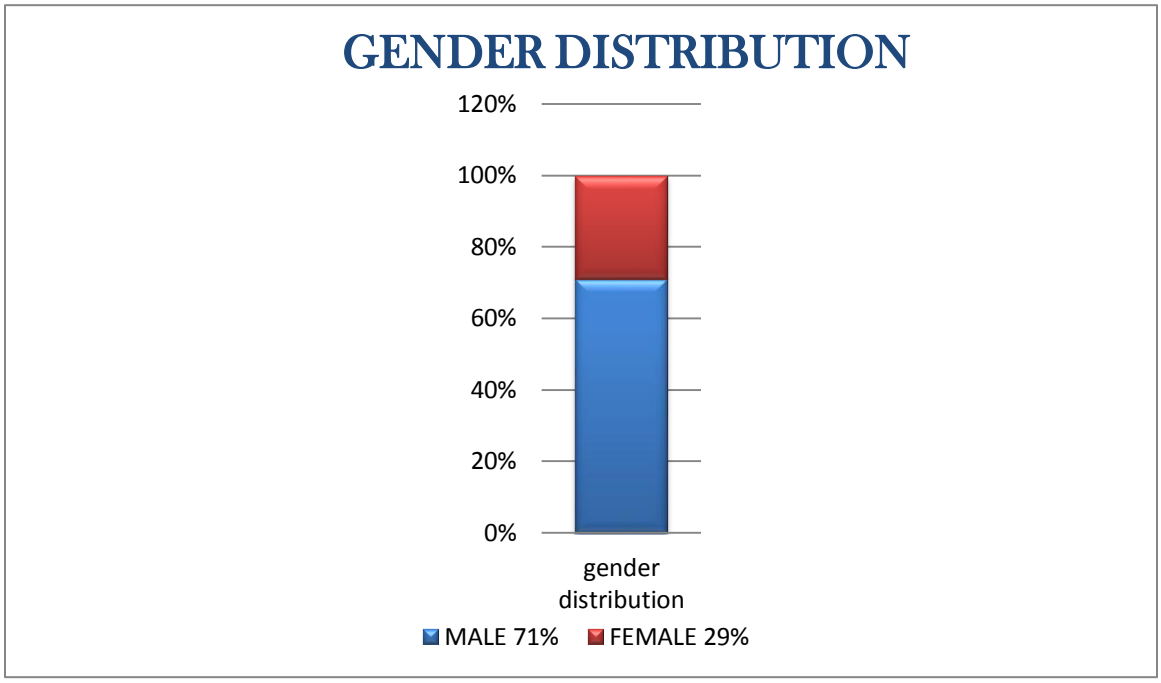
Data Collected:

Preoperative demographic and clinical data, details of the surgical procedure, postoperative course, and complications were collected prospectively. Various presentations of tubercular abdomen along with the procedure done were analyzed. Post operatively patients were followed up to look for development of complications. All the patients were discharged after starting ATT. Complications included
wound infection, development of adhesions, anastomotic leak, respiratory complications, duration of hospital stay.

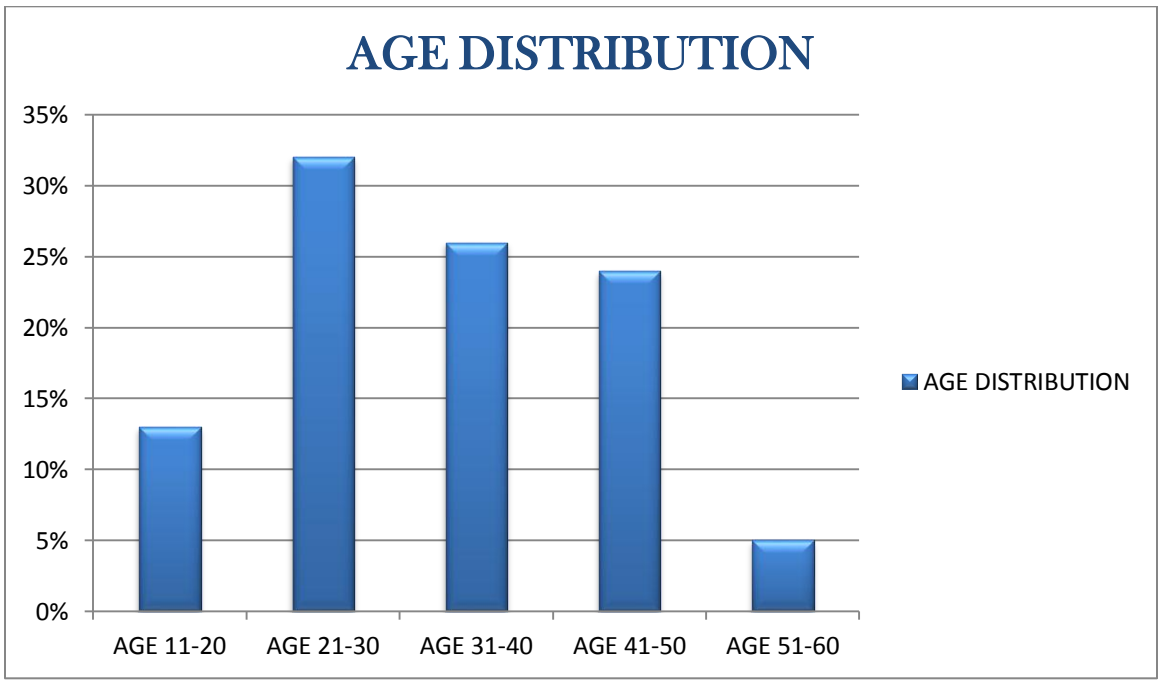
Results:

- Sixty two cases of acute abdomen were taken up for study.
- All cases were taken up for emergency laparotomy on the day of admission
- Stricture of ileum was the most common presentation in emergency followed by ileo cecal tuberculosis (46%)
- Most commonly performed procedure was resection anastomosis (31%)
- All the patients were started on ATT on a average of 15 th post operative day.

The following observations were made.....



Of 62 patients 44 were male and 18 were female, which were 71% and 29% respectively.



Among the patients studied, 20 were from the age group of 21-30yrs corresponding to 32%.

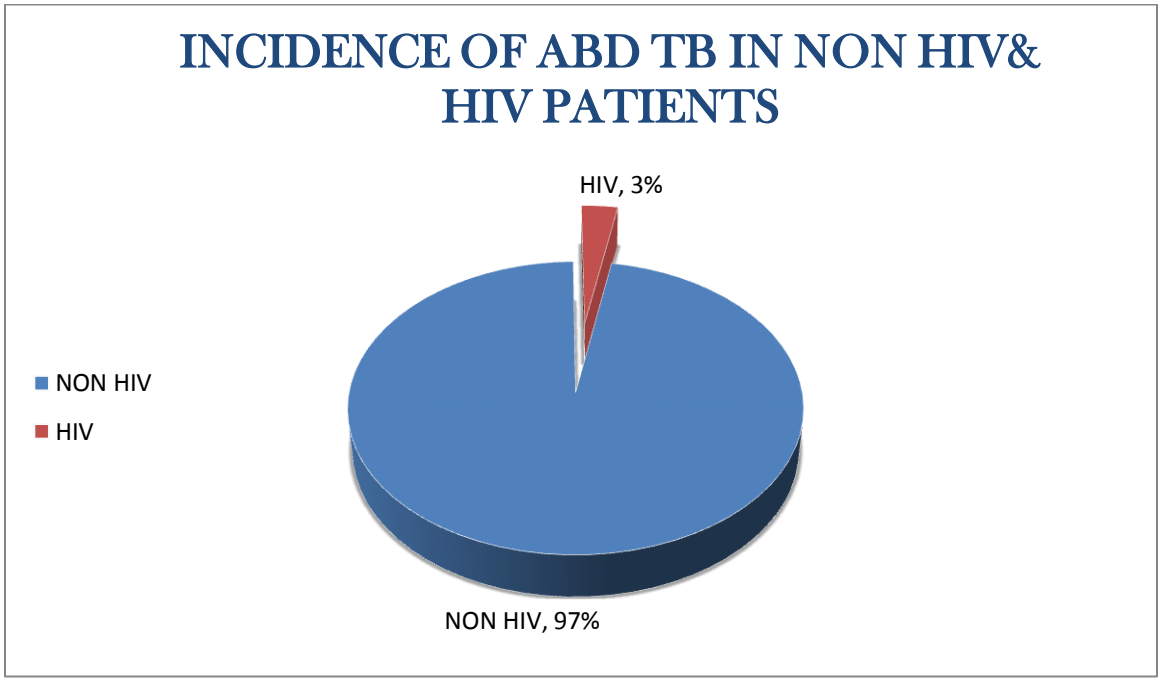
Age group 11-20= 8(13%)

Age group 21-30=20(32%)

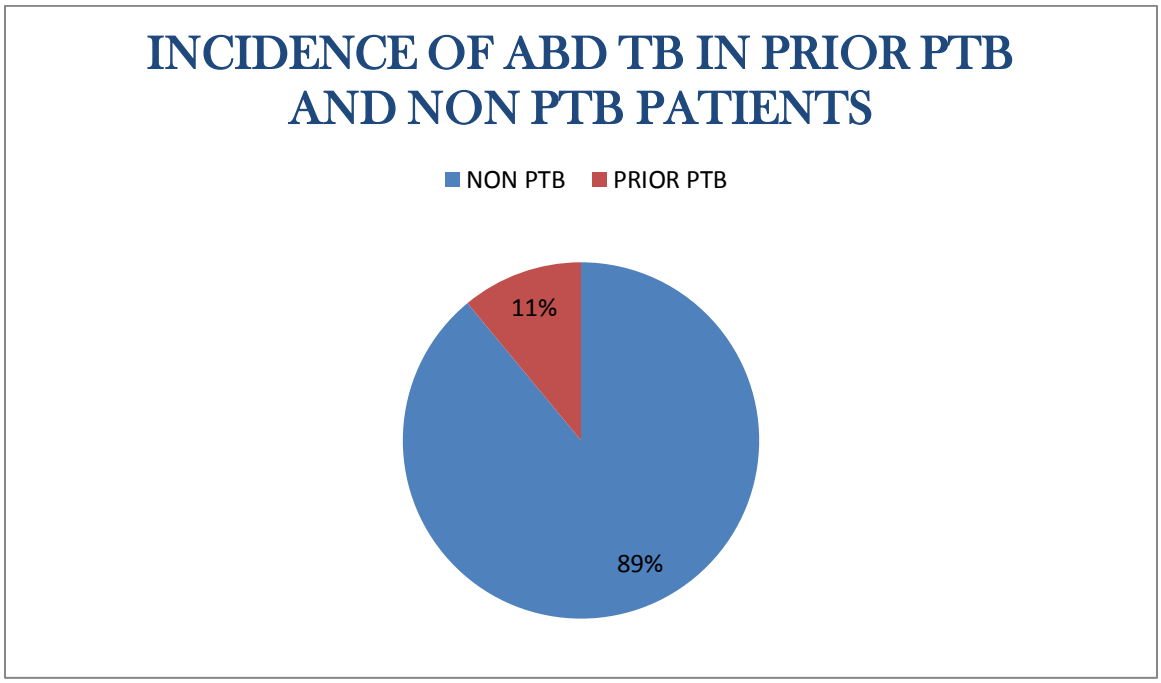
Age group 31-40=16(26%)

Age group 41-50=15(24%)

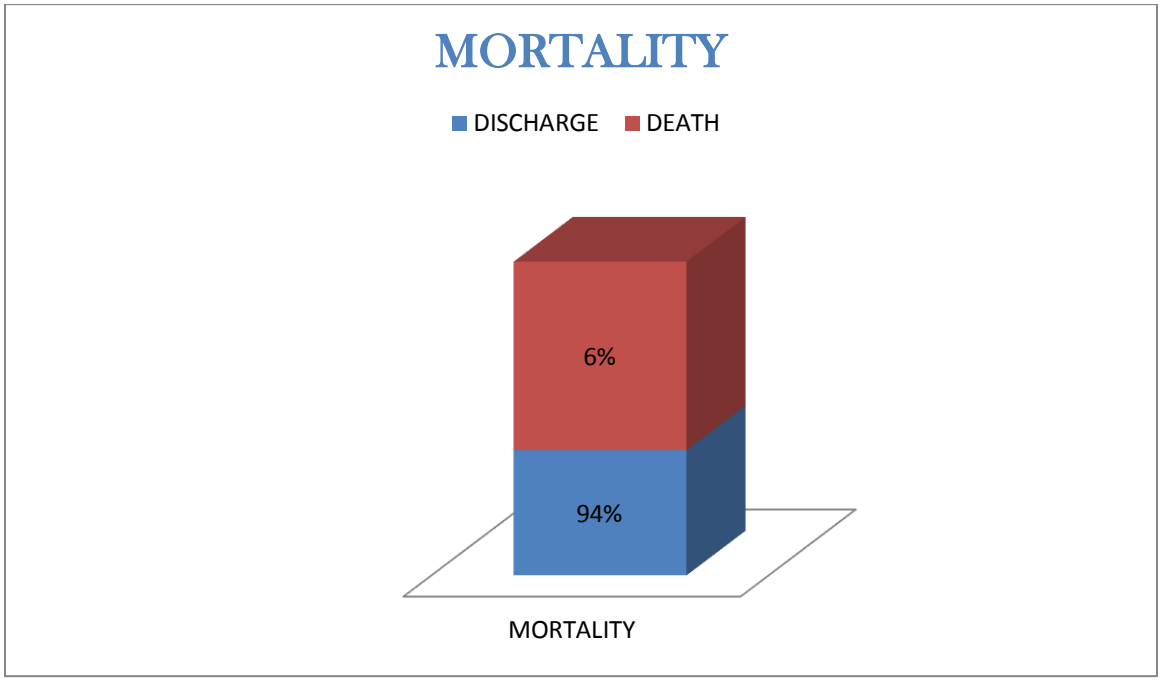
Age group 51-60=3(5%)



Among the patients, only 2 had HIV infection, making it about 3% of total.

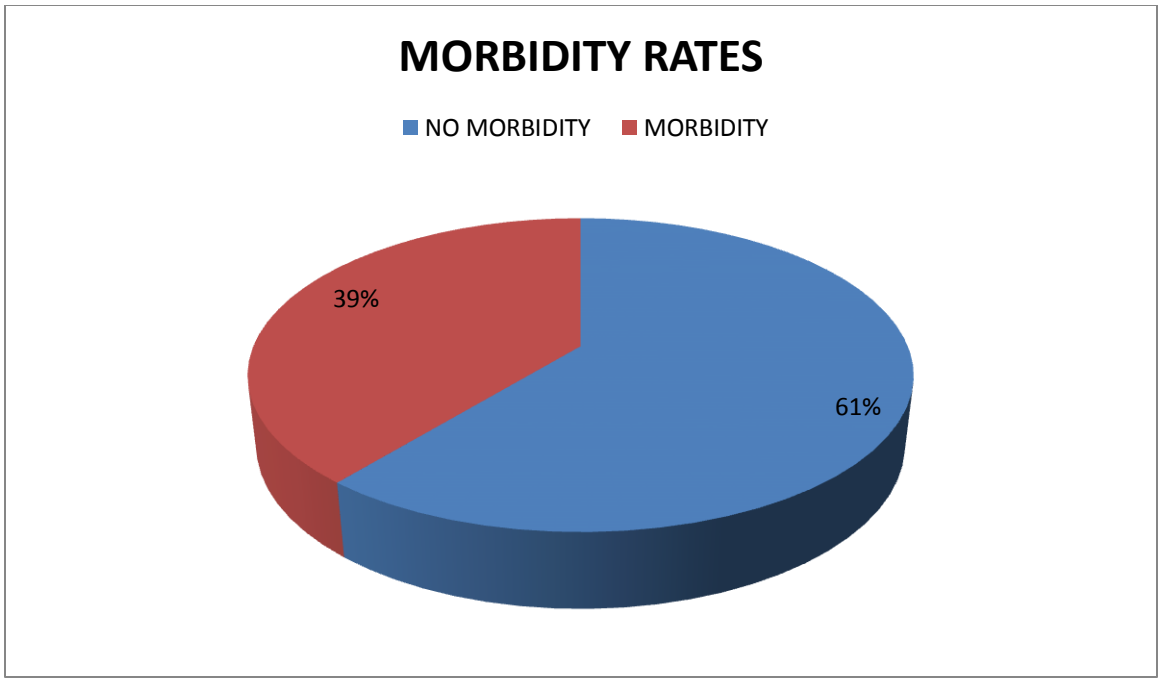


7 patients had h/o of pulmonary tuberculosis previously, who had completely finished their ATT.



Mortality among the treated patients were 6%(4 patients).

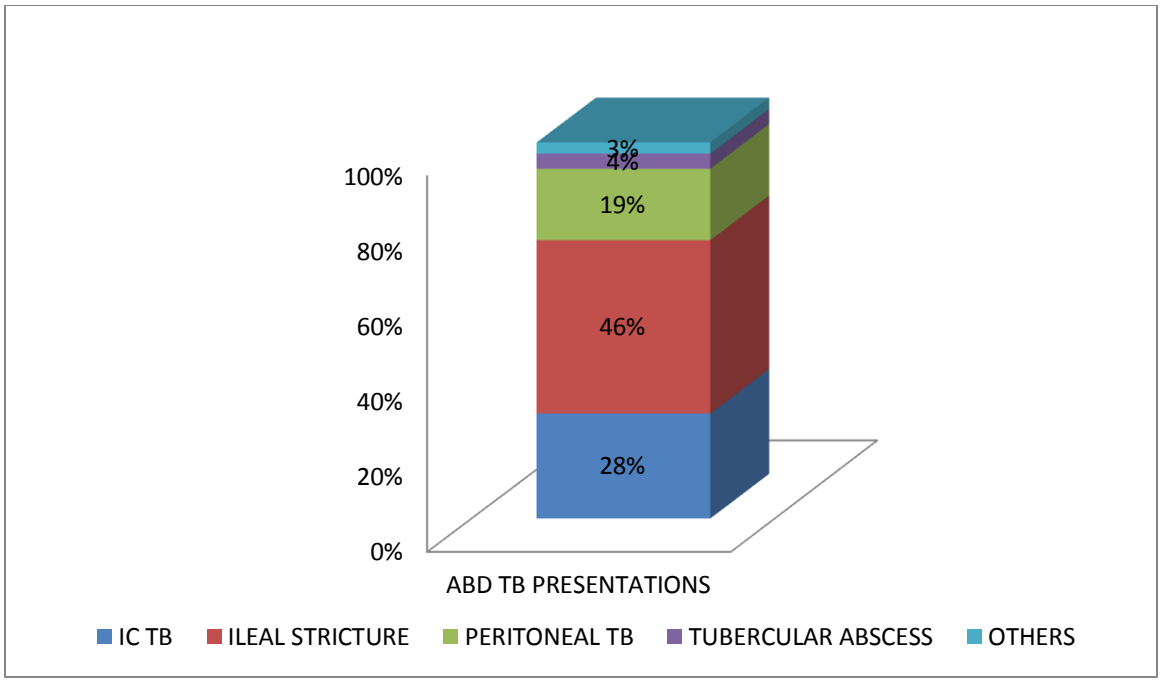
All the deaths were due to septicemia.



Morbidity was 39% (24 patients)

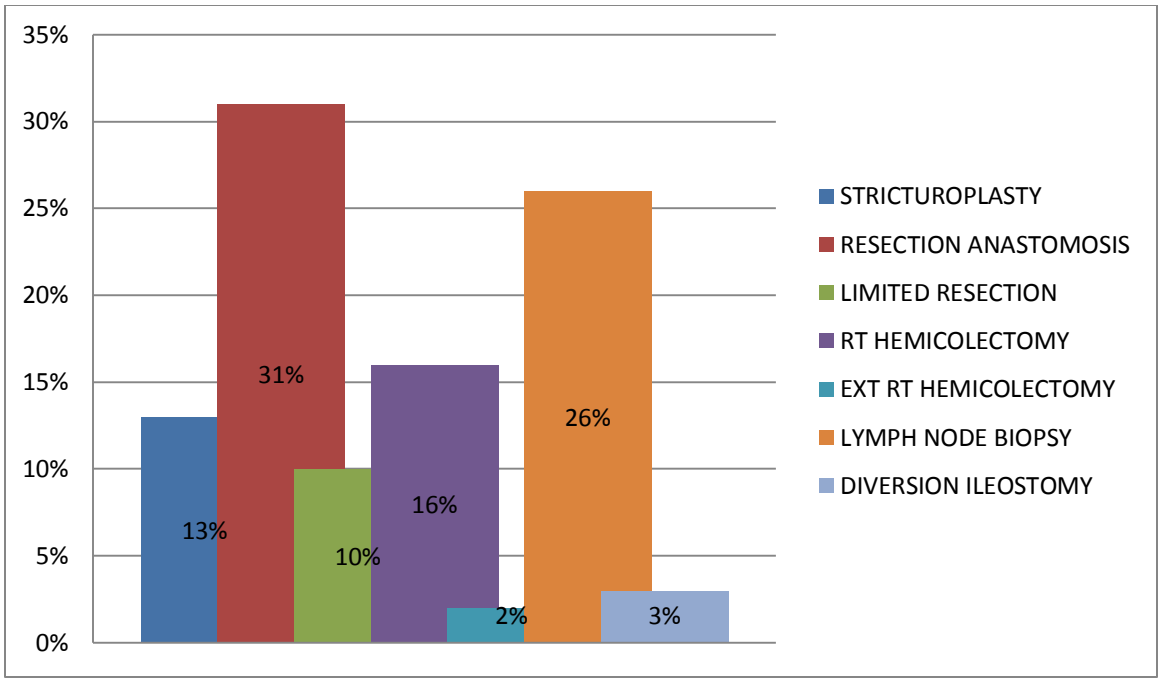
Most common factor responsible for morbidity was wound infection.

Other complications included respiratory tract infection and anastomotic leak.



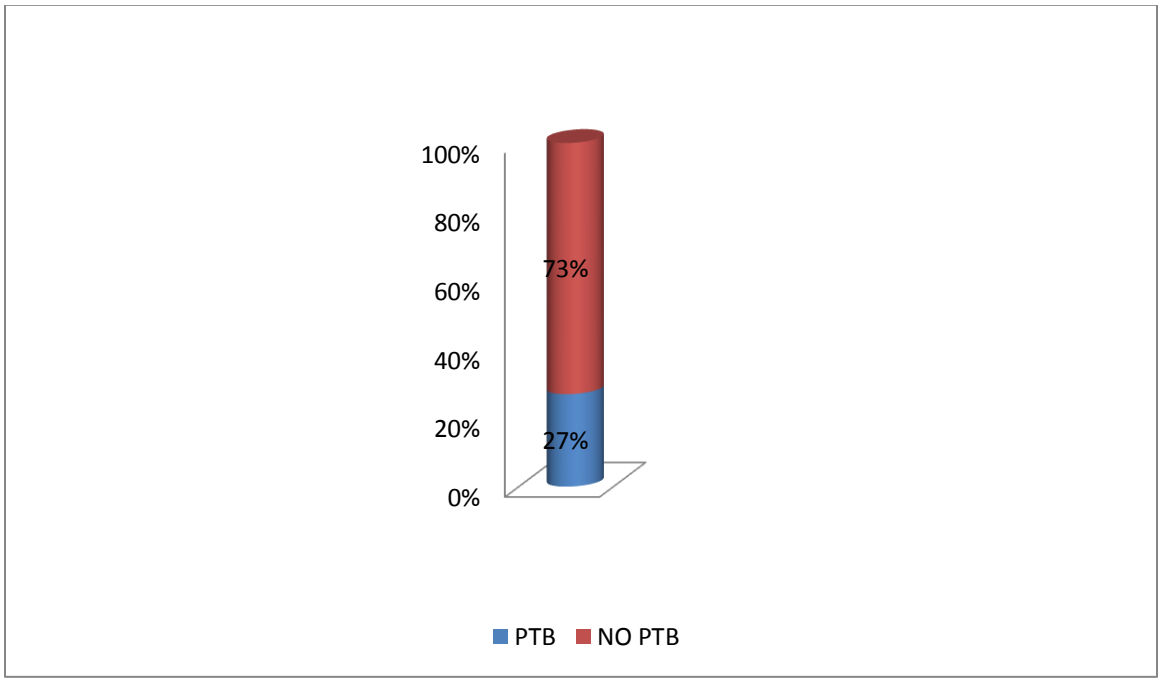
Various presentations of tubercular abdomen were

- Ileo ceacal tuberculosis =17(28%)
- Ileal Stricture =28(46%)
- Peritoneal tuberculosis =12(19%)
- Tubercular Abscess =3(4%)
- Other presentations =2(3%)
    - Transverse colon stricture=1
    - Retroperitoneal Lymphadenopathy=1

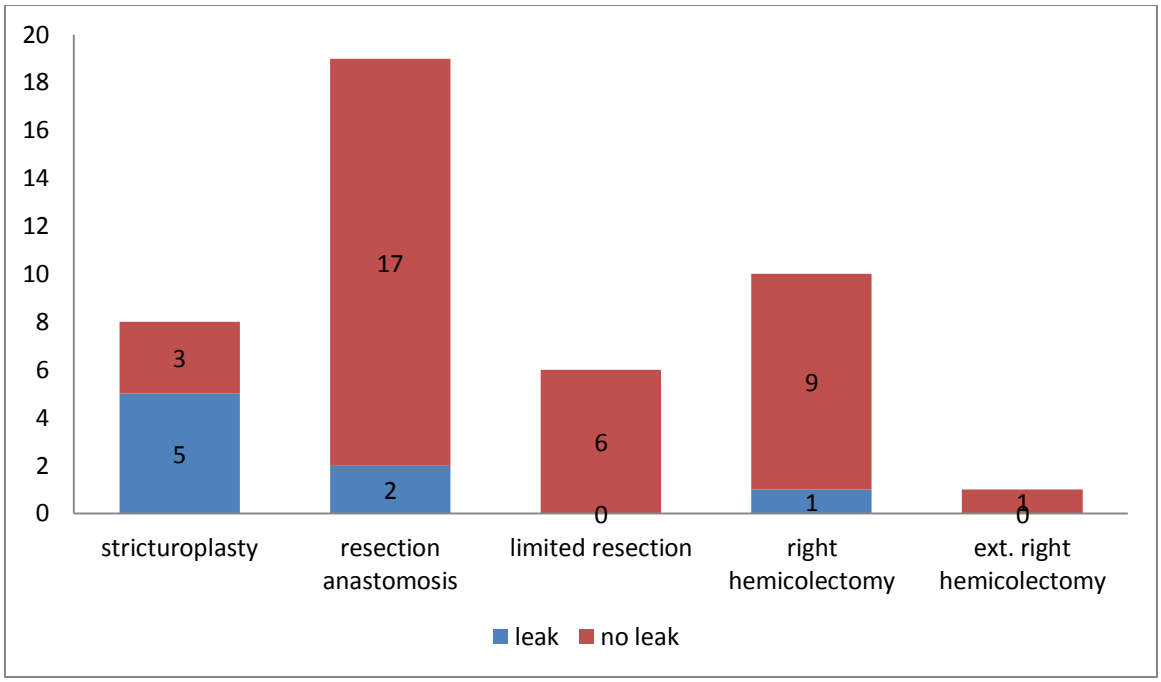


Surgical interventions performed were,

- Stricturoplasty =8(13%)
- Resection Anastomosis of Ileal stricture with/without proximal diversion ileostomy =19(31%)
- Limited Resection of Ileum, Caecum & Ascending Colon with Ileo-Ascending anastomosis with/without proximal diversion ileostomy =6(10%)
- Right Hemicolectomy with/without proximal diversion ileostomy =10(16%)
- Extended Right Hemicolectomy =1(2%)
- Lymph Node biopsy =16(26%)
- Diversion ileostomy =2(3%)



Of the patients diagnosed with abdominal tuberculosis about 17(27%) had evidence of pulmonary tuberculosis. Among the patients diagnosed with PTB, 2 out of 17 had history of prior tuberculosis.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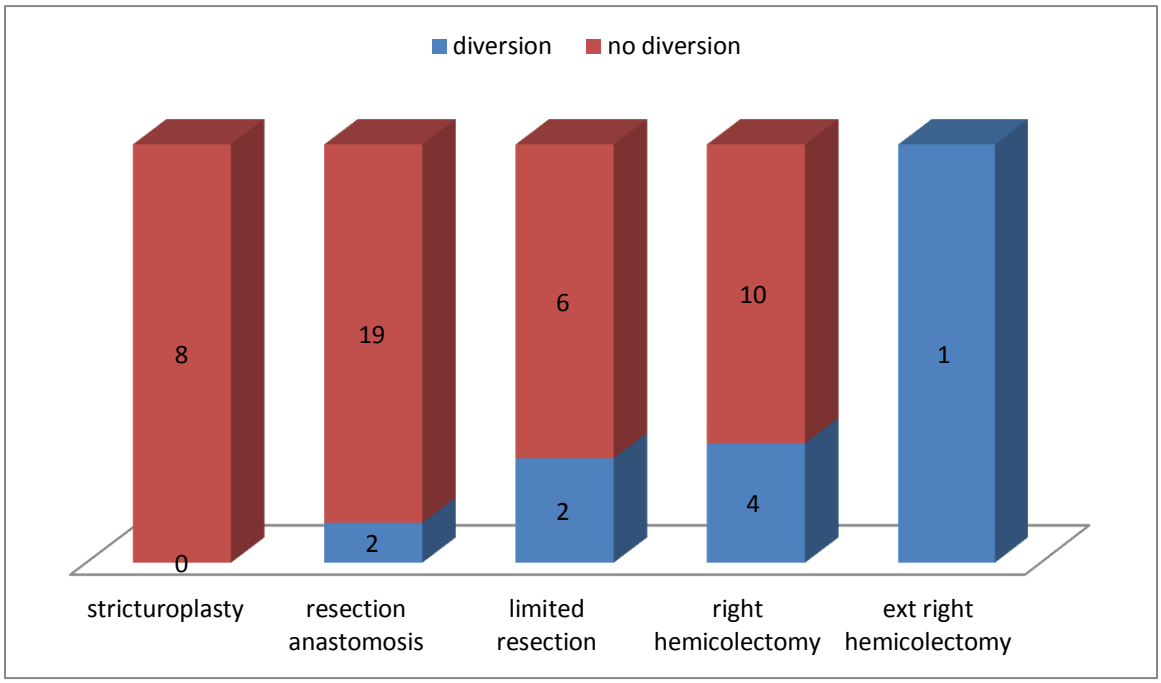
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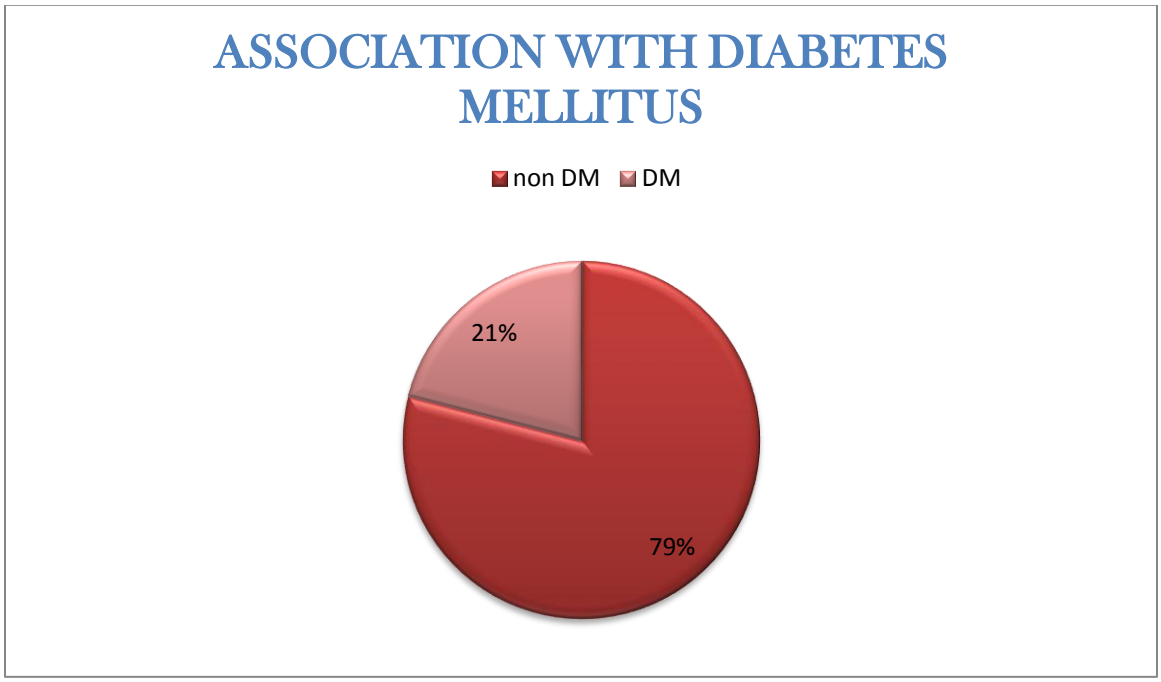

Diabetes mellitus was present in 21% of the patients(13 patients).

Discussion:

Age Distribution:

Among the patients studied, majority of abdominal tuberculosis was present in the age group of 21-30 yrs (32%)

This is followed by higher incidence among 31-40 yrs(26%)

Most common age group affected by PTB belong to 21-40 yrs, the so called working age group.

Our study of Abdominal tuberculosis also reports the same age wise distribution, reflecting the same as in the distribution of Pulmonary TB.

Gender Distribution:

Male patients were majority (71%)

Females(29%)

Male female ratio is 3:1.

Socio economic Distribution:

Majority of the disease is among lower socio economic group (83%).

This reflects the overall incidence of tuberculosis.

History of prior tuberculosis:

Of the 62 patients presented,

7(11%) had previous history & treatment for pulmonary Tuberculosis.

All the patients had complete course of ATT for PTB and there was no default or relapse.

Among the 62 patients presented about 35 patients had prior history of abdominal pain, which were treated without any approximate investigations. This is about 22%.

Among these patients,(35) about 10 were found to have PTB post operatively, about 30%, who were diagnosed with sputum smear for AFB.

Presumptive treatment of these patients would have decreased the incidence of abdominal tuberculosis also in this sub group of patients($p<0.05$).

PTB diagnosed after surgery :

After surgery, 17(27%) patients were found to have pulmonary tuberculosis.

All these patients underwent CXR-PA view, sputum for AFB.

15 of the patients were newly diagnosed with pulmonary tuberculosis.

2 of the patients previously had PTB.

Abdominal tuberculosis is present as a single entity in majority of the patients(73%) in our study group, without any pulmonary involvement.

Though coexisting PTB was present in a relatively small proportion of patients(27%), the presence of PTB in them indicates a more disseminated form of .disease, requiring intensive follow up.

Types of presentations:

- In our study, Ileal stricture was the most common presentation.
- Though literature says ileo caecal tuberculosis is the most common type, our study analyzed only the acute presentations, where obstruction was the most common clinical presentation.
- Two rare types of presentations, such as transverse colon stricture and retro peritoneal lymphadenopathy were also seen.
- Even for the strictures, we preferred resection anastomosis than stricturoplasty, as the disease is an ongoing process.
- Resection anastomosis was the most frequently performed procedure for strictures.
- They have the advantage of removing the entire diseased portion of the small bowel, when compared to stricturoplasty, where the diseased portion remains.

- Not only it has the advantage of removing the disease, but also the decrease in the complications like anastomotic leak (impaired healing in the stricturous portion) and recurrence of the obstruction (Also the the ATT regimen would have increased the inflammation thereby causing subsequent fibrosis and might have lead to obstruction again)

Morbidity:

Wound infection was the most common postoperative complication, causing increased morbidity.

Patients newly diagnosed as having PTB had increased incidence of respiratory complication. These respiratory complications include, pneumonia, pleural effusion, fall of saturation making the patient dependent on a mechanical ventilatory support.

These increase in respiratory complications in new PTB patients can be attributed to the active disease.

All these patients were given appropriate antibiotics after performing wound swab, sputum culture& sensitivity.

Anastomotic leak which is normally at a rate of 2- 4 % was relatively high among our patients, (about 25%).

Patients, who underwent stricturoplasty had very high rates of leak, about 62.5%.

Even for the other procedures, it was significantly high, resection anastomosis(10.5%) & Right Hemicolectomy(10%).

The performance of proximal diversion, significantly reduced the complication due to anastomotic leak.

Proximal diversion was performed in about,

11% (2/19) of resection anastomosis (non diversion leak rate was 11%),

33% (2/6) of limited resection group (non diversion leak rate 0%),

40% (4/10) of right hemicolectomy group (non diversion leak rate 10%) &

100% (1/1) of ext.right hemicolectomy group (non diversion leak rate 0%).

Mortality:

Of the patients operated, 4 patients died. All the death were due to septicemia. It is about 6% of the total patients studied.

S. NO	Name	Age/sex	Co morbidity	shock	Symptoms - dur	diagnosis	Procedure done	Cause of death
1.	THANGAVEL	39/M	-	-	3 DAYS	ILE.STRIC	RESEC ANAS	ANAS. LEAK/SEPTICEMIA
2.	KOTTI	46/M	-	-	4 1/2 DAYS	IC TB	DIV. ILEO	SEPTICEMIA
3.	KAMALA	34/F	-	+	2 DAYS	IC TB/DIL SMA BOW LOOPS	RT HEMIC0LEC / DIV ILEO	SEPTICEMIA
4.	KARUPANNAN	27/M	-	+	2 DAYS	ILE. STRIC	RESEC ANAS	SEPTICEMIA

Post operative ATT:

All the patients underwent postoperative evaluation for pulmonary tuberculosis.

The investigations performed were, CXR PA view, sputum smear for AFB & sputum culture for AFB.

On average, ATT was started on 16 th pod.

All the patients were treated with Cat I ATT.

The problem with Extra pulmonary tuberculosis is the difficulty in finding whether the patients had MDR tuberculosis. Nowadays, with the incidence of MDR TB on rise, chances of an Extrapulmonary TB to be a drug resistant one are very high.

Conclusion:

- Ileal stricture was the most common presentation of the acute abdominal tuberculosis.
- Prior thorough investigations for Pulmonary Tuberculosis with sputum AFB in patients chronic abdominal pain and early initiation of the treatment will definitely decrease the progression into abdominal tuberculosis a significant number of patients.
- Performing a proximal diversion, along with the definitive procedure, significantly decreases the incidence of anastomotic leaks.

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ANNEXURE A:

ABBREVATIONS USED:

PERI.TB	PERITONEAL TUBERCULOSIS
PTB	PULMORARY TUBERCULOSIS
RIF	RIGHT ILIAC FOSSA
TEND	TENDERNESS
PRO	PROBE
MES	MESENTERIC
BX	BIOPSY
ATT	ANTI TUBERCULOSIS TREATMENT
POD	POST OPERATIVE DAY
FOLL	FOLLOW
COMPL	COMPLICATIONS
EXAM	EXAMINATION
CO.MOR	CO MORBIDITY
PRE	PREVIOUS
FIND	FINDING
PROD	PROCUDURE
ABD	ABDOMEN
DIST	DISTENSION

BS	BOWEL SOUND
DIL	DILATED
SMA	SMALL
BOW	BOWEL
ILEA	ILEAL
STRIC	STRICTURE
IC	ILEOCAECAL
PROX	PROXIMAL
M	MALE
F	FEMALE
GUAR	GUARDING
RIG	RIGIDITY
F.F.	FREE FLUID
PERIT	PERITONEAL
C/S	CULTURE AND SENSITIVITY
OMEN	OMENTUM
BIOP	BIOPSY
RTI	RESPIRATORY TRACT INFECTION
MAL	MALIGNANCY
IL	ILEUM

HEM COLEC	HEMICOLECTOMY
STRIC PLAST	STRICTUROPLASTY
PERF	PERFORATION
PERIT	PERITONEAL
APPE	APPENDICULAR
OBS	OBSTRUCTION
YRS	YEARS
RESEC	RESECTION
ANAS	ANASTAMOSIS
LIM	LIMITED
FLU	FLIUD
ANAL	ANALYSIS
MIN	MINIMAL
ASCIT	ASCITIS
EVAL	EVALUATION
DIV	DIVERSION

ANNEXURE B:

MASTER CHART:

S.NO	NAME	AGE	SEX	I.P.NO:	PREV HISA BD.PAIN	SYMPTOMS	EXAM	CO.MOR	INV		PRE.TB	ATTHIS	HIV	FIND	PRO	COMPL	FOLL.UP	
									USG	CT							ATT	POSTOPPTB
1.	ANANDHAN	32	M	94597	+/6MON	FEVER, ABD. PAIN- 10 DAYS	RIF TEND	-	RT. IL F PRO TEND	-	-	-	-	MES. DISEASE	MES.NODE BX	-	ATT ON 14 POD	
2.	RAJSEKAR	36	M	97000	+/2MON	ABD. DIST – 21/2 DAYS	DIST. ABD IBS	-	DIL. SMA BOW	SMA BOW OBS	-	-	-	ILE. STRIC	STRIC PLASTY	ANAS LEAK	ATT ON 14 POD	
3.	MANOHAR	48	M	34002	-	ABD. DIST- 2 DAYS	DIST. ABD N BS	SHT	DIL. SMA BOW	SMA BOW OBS ? IC TB	YES	5 YRS	-	IC TB WITH DIL PROX. SMA BOW	LIMITED RESEC ANAS	WOUND GAPING/ RTI	ATT ON 20 POD	+
4.	NAGAPPAN	55	M	89186	+/1YR	FEVER,ABD. PAIN- 2 DAYS	GUAR /RIG +	SHT	F.F ABD	MES. NOD/F. F ABD	-	-	-	PERIT ABSCESS	PUS C/S MES. NODE/ OMEN BIOP	WOUND INFECTION	ATT ON 21 POD	
5.	DEIVANAI	60	F	76248	+/1YR	ABD. PAIN&DIST 31/2 DAYS	RIF MASS DIST/ GUAR /DIS +	DM/SHT	RIF MASS/DIL SMA BOW ?TB/? MAL	RIF MASS/ DIL SMA BOW ?TB/? MAL	-	-	-	IC MASS WITH PROX. SMA BOW	RT HEMICOLEC WITH PROX IL DIVER	-	ATT ON 14 POD	+
6.	MUNUSAMY	42	M	100128	-	FEVER/ABD. PAIN/ABD. DIST 2 DAYS	ABD TEND/ DIST/	-	DIL.SMA.BO WPRO TEND	-	-	-	-	ILE.STRIC WITH MES NODES	STRIC PLAST/MES NODE BX	-	ATT ON 14	

							DBS										POD	
7.	LAXMI	22	F	64128	+/N.K	FEVER/ABD.PAIN 1½ DAYS	ABD TEND/GUAR/RIG	-	PRO.TEND/F.F ABD	-	-	-	-	ILE. STRIC/PROX PERF/ PERIT CONTAM	RESEC ANAS/ PROX IL DIVER	-	ATTON 14 POD	
8.	MANI	46	M	21346	+/3MON	ABD.DIST 3DAYS	ABD DIST/DBS	-	DIL.SMA BOW	IC MASS ? MALIG/ ?TB	-	-	-	IC MASS WITH PROX. SMA BOW	RT HEMICOLECT	ANAS. LEAK	ATTON 20 POD	+
9.	YUVARAJAN	18	M	17431	+/1MON	FEVER/ABD PAIN 7 DAYS	LOWER ABD GUAR/RIGID	-	RIF PRO TEND/APPE MASS	-	-	-	-	MES.DISEASE	PERIT BX/MES.NODE BX	-	ATTON 14 POD	
10.	RAMAN	29	M	13420	-	ABD.DIST/PAIN 2 DAYS	ABD. DIST/DBS	-	DIL.SMA	SMA BOW OBS	YES	14 YRS	-	ILE.STRIC	RESEC ANAS	-	ATTON 14 POD	+
11.	SAMSUDEEN	41	M	9023	-	ABD.DIST/PAIN 2 1/2 DAYS	ABD. DIST/DBS	-	DIL.SMA	SMA BOW OBS	-	-	-	ILE.STRIC	STRIC PLAST/MES NODE BX	ANAS LEAK	ATTON 14 POD	
12.	YUSUF	30	M	24782	+/2MON	FEVER,ABD. PAIN- 1 1/2 DAYS	ABD. DIST/DBS	-	DIL.SMA	SMA BOW OBS	-	-	-	ILE.STRIC	RESEC ANAS	-	ATTON 14 POD	+
13.	LOGANATHAN	29	M	27145	+/1MON	FEVER/ABD. PAIN/ABD. DIST 2 DAYS	ABD TEND/DIST/DBS	-	DIL.SMA.BOWPRO TEND	?STRICT SMA BOW/PROX OBS	-	-	-	IC TB/ PROX. SMA BOW OBS	LIM. RESEC	-	ATTON 14 POD	
14.	HEMAVATHY	35	F	30986	-	FEVER/ABD. PAIN/DIST 3 DAYS	ABD TEND/DIST/DBS	-	DIL BOW LOOPS	SMA BOW OBS	-	-	-	ILE.STRIC	STRIC PLAST/MES NODE BX	-	ATTON 14 POD	
15.	MALINI	24	F	45693	+/1MD	ABD. DIST 2 ½ DAYS	ABD TEND/DIST/DBS	-	DIL BOW LOOPS	DIL BOW LOOPS	YES	10 YRS	-	ILE.STRIC	RESEC ANAS	-	ATTON 14 PO	

					N												D	
16.	SEKARAN	37	M	48923	+/3MON	FEVER/ABD. PAIN/ABD. DIST 2 DAYS	ABD TEND/DIST/DBS	-	RIF PRO TEND/APPE MASS	IC MASS ? MALIG/ ?TB	-	-	-	IC TB/ PROX. SMA BOW OBS/PERITON CONTAM	LIM. RESEC	-	ATTON 14 POD	
17.	RAJENDIRAN	43	M	57892	+/2MON	FEVER/ABD. PAIN/ABD. DIST 4 DAYS	ABD TEND/DIST/DBS	-	DIL BOW LOOPS	DIL BOW LOOPS	YES	3 YRS	-	IC TB/ PROX. SMA BOW OBS/	LIM. RESEC	WOUND GAPING/ RTI	ATTON 14 POD	+
18.	REVATHY	34	F	42157	+/3MON	ABD PAIN/ FEVER 4 DAYS	GUARD/RIGID +	-	PROB TEND/F.F	F.F.	-	-	-	PERIT ABSCESS	PUS C/S MES. NODE/ OMEN BIOP	WOUND INFECTION	ATTON 24 POD	
19.	MUNIYAN	47	M	45221	+/4MON	FEVER/ABD. PAIN/ABD. DIST 4 DAYS	ABD DIST/DBS	SHT	PROB TEND/F.FDIL SMA BOW	F.F.	-	-	-	IC TB WITH PROX. SMA BOW/ASCIT	RT HEMICOLECT /ASCIT FLU ANAL	-	ATTON 14 POD	
20.	THANGAVEL	39	M	36771	-	ABD PAIN/ABD DIST 3 DAYS	ABD DIST/DBS	-	DIL.SMA BOW	DIL BOW LOOPS	-	-	-	ILE.STRIC	RESEC ANAS	ANAS. LEAK/SEPT/DEATH		
21.	DAMODHARAN	40	M	43612	-	FEVER/ABD. PAIN/ABD. DIST 2 ½ DAYS	ABD DIST/DBS	-	DIL.SMA BOW	DIL BOW LOOPS	-	-	-	ILE.STRIC	STRIC PLASTY	-	ATTON 14 POD	+
22.	SUNDARI	46	F	57213	+2MON	FEVER/ABD PAIN ABD DIST 3 DAYS/	ABD DIST/DBS	-	PROB TEND/ DIL SMA BOW	DIL BOW LOOPS	-	-	-	IC TB/DIL SMA BOW LOOPS	RT HEMICOLECT /ASCIT FLU ANAL	WOUND INFECTION	ATTON 18 POD	
23.	GOMATHI	43	F	47117	+/ 11/2MON	ABD PAIN/FEVER	RIF TEND/MIN RIF GUARD	-	PROB TEND	?APP ABSCESS	-	-	-	MES. TB/ MIN ASCIT	MES.NODE BX/ASCT ANAL	-	ATTON 16 POD	+
24	SARAVANAN	27	M	37169	-	FEVER/ABD PAIN ABD DIST	ABD DIST/	-	DIL.SMA BOW	DIL BOW	-	-	-	ILE.STRIC/ MES TB	STRIC PLASTY/MES.	ANAS.LEAK /WOUND	ATTON 19	

.						4 DAYS	DBS			LOOPS					NODE BX	INFECTIO N	PO D	
25.	HARIHARAN	17	M	68711	+/3 YRS	ABD PAIN/ABD DIST 2 2/2 DAYS	ABD DIST/ IBS	-	PROB TEND/DIL.SMA BOW	DIL BOW LOOPS	YES	5 YRS	-	ILE.STRIC	RESEC ANAS	-	ATT ON 14 PO D	
26.	MICHAEL	36	M	57357	-	FEVER/ABD. PAIN/ABD. DIST 23½ DAYS	ABD DIST/ ABS	-	PROB TEND/ DIL SMA BOW	-	-	-	-	ILE STRIC/ DIL SMA BOW / PERITON	RESEC ANAS/PROX DIVER	-	ATT ON 25 PO D	
27.	PANDIAN	39	M	12901	-	FEVER/ABD. PAIN2 DAYS	ABD TEND/ GUAR D	-	ASCT FOR EVAL.	E/O REE FLUID	YES	12 YRS	-	ASCIT / MES. TB	ASCIT EVAL/ MES NODE BX	-	ATT ON 14 PO D	
28.	SUBRAMANIAN	57	M	45190	+/5 MON	ABD DIST/ABD PAIN 4 DAYS	ABD TEND/ ABD DIST	DM	?CECAL MALIG	?GROW TH CECUM	-	-	-	IC GROWTH/DIL SMA BOW	RT HEMICOLEC/ PROX SMA DIV	-	ATT ON 30 PO D	+
29	RASHEED	22	M	23145	+/3 ½ MON	ABD DIST/ABD PAIN 2 DAYS	ABD TEND/ ABD DIST	-	DIL.SMA BOW	DIL BOW LOOPS	-	-	-	ILE STRIC/ DIL SMA BOW /	RESEC ANAS/PROX DIVER	-	ATT ON 14 PO D	-
30.	RAMACHANDRAN	48	M	56123	-	FEVER/ABD PAIN ABD DIST 3 DAYS/	ABD DIST/ OBS	-	PROB TEND/ DIL SMA BOW	DIL BOW LOOPS	-	-	-	ILE STRIC/ DIL SMA BOW / PERITON	RESEC ANAS		ATT ON 14 PO D	
31.	THANGARAJ	30	M	24178	+/2 MON	ABD PAIN/ABD DIST 21/2 DAYS	RIF MASS ABD DIST/ D BS	-	RIF MASS FOR EVAL	RIF MASS FOR EVAL/ SMA BOW OBS	YES	3 YRS	-	IC TB	LIM RESEC/ DIV ILEO	-	ATT ON 16 PO D	
32.	MUNIYAMMA	45	F	119803	-	FEVER/ABD PAIN 3 DAYS	MIN DIFF GUAR D/RIG ID	-	PROB TND	-	-	-	-	MES TB	MES NODE BX	-	ATT ON 12 TH DAY	+
33	CHANDRAN	3	M	21478	+/	ABD PAIN/ABD	ABD DIST/	-	DIL SMA	DIL SMA BOW/?	-	-	-	ILE STIRC	RESEC ANAS	-	ATT ON	

.		3			1 M O N	DIST 2 DAYS	N BS		BOW	STIR							12 TH DAY	
34.	POORNIMA	29	F	67129	+ / 3 M O N	ABD PAIN/ABD DIST 3 ½ DAYS	ABD DIST/ DBS	-	DIL SMA BOW	-	-	-	-	ILE STIRC	STRIC. PLAST	-	ATT ON 12 TH DAY	
35.	KOTTI	46	M	58267	-	FEVER/ ABD PAIN/ABD DIST 2 ½ DAYS	ABD DIST/ NBS	-	DIL SMA BOW	DIL SMA BOW/? STIR	YES	4 YRS	YES	IC TB	DIV. ILEO	SEPT/DEATH		+
36.	VALLI	30	F	19245	-	FEVER/ABD PAIN/ABD DIST 4 ½ DAYS	ABD DIST/ DBS/ GUAR RIGID	-	DIL SMA BOW/ IC MASS	RIF MASS FOR EVAL/ SMA BOW OBS	-	-	-	IC TB	LIM RESEC/ DIV ILEO	WOUND INFECTION	ATT ON 24 TH DAY	
37.	SASIKUMAR	28	M	102381	+ / 4 M O N	ABD PAIN/ABD DIST3 ½ DAYS	ABD DIST/ DBS	-	PROB TEND/ DIL SMA BOW	DIL BOW LOOPS	-	-	-	ILE STIRC	RESEC ANAS	-	ATT ON 20 POD	+
38.	PONNI	41	F	73417	-	ABD PAIN/ABD DIST 1 DAY	ABD DIST/ I BS	-	DIL SMA BOW	-	-	-	-	ILE STIRC	RESEC ANAS	-	ATT ON 14 POD	
39.	SELVAM	30	M	47223	+ / 1 Y R	FEVER/ABD PAIN/ABD DIST 4 DAYS	ABD DIST/ DBS	-	DIL SMA BOW	DIL BOW LOOPS	-	-	-	ILE STIRC	STRIC. PLAST	ANAS LEAK	ATT ON 18 POD	
40.	ANANDHAN	45	M	71905	+ / 1 ½ M O N	FEVER/ABD PAIN 2 DAYS	DIFF GUAR D RIGID	DM	PROB TND	N	-	-	-	MES. TB/ MIN ASCIT	MES NODE BX	-	ATT ON 14 POD	
41.	SHANMUGAM	57	M	89157	+ / 5 M O N	FEVER/ABD PAIN 2 ½ DAYS	DIFF GUAR D RIGID	-	PROB TND	N	-	-	-	MES. TB/ MIN ASCIT	MES NODE BX	-	ATT ON 18 POD	+

42.	KAMALA	34	F	56027	-	ABD DIST 2 DAYS	ABD DIST/ DBS	-	RIF MASS FOR EVAL/ SMA BOW OBS	CECAL MASS /PROX SMA BOW OBS	-	-	-	IC TB/DIL SMA BOW LOOPS	RT HEMICOLEC/ DIV ILEO	SEPT/DEATH		
43.	CHINNASAMY	37	M	19842	-	FEVER/ABD PAIN/ABD DIST 1 DAY	ABD DIST/ DBS	-	? RIF MASS EVAL	-	-	-	-	IC TB/DIL SMA BOW LOOPS	LIM RESEC	WOUND INFECTION/RTI	ATTON 14 POD	
44.	MARIAPOOSANAM	29	M	2315	-	ABD PAIN/ABD DIST 1½ DAYS	ABD DIST/ DBS	-	DIL SMA BOW	DIL SMA BOW	-	-	-	ILE STIRC	STRIC. PLAST	ANAS. LEAK	ATTON 20 POD	
45.	SUNDARAMBAL	43	F	52310	+/ 2½ MON	ABD PAIN/ABD DIST 4 DAYS	ABD DIST/ DBS	-	DIL SMA BOW	DIL SMA BOW	-	-	-	ILE STIRC	RESEC ANAS	-	ATTON 14 POD	
46.	CHELLAPPAN	39	M	50115	+4 MON	ABD PAIN/ABD DIST 3 ½ DAYS	ABD DIST/ DBS	-	PROB TEND/DIL SMA BOW	? IC MASS/ SMA BOW OBS	-	-	-	IC TB/DIL SMA BOW LOOPS	RT HEMICOLEC	WOUND INFECTION	ATTON 16 POD	
47.	THANIGAIMANI	22	M	60338	+/ 8 MON	ABD PAIN/ABD DIST 4 ½ DAYS	ABD DIST/ DBS	-	DIL SMA BOW	DIL SMA BOW				ILE STIRC	RESEC ANAS	-	ATTON 24 POD	+
48.	MUMTAJ	30	F	1907	-	FEVER/ABD PAIN/ABD DIST 3 DAYS	ABD DIST/ DBS	-	RIF MASS FOR EVAL/ SMA BOW OBS	-	-	-	-	IC TB/DIL SMA BOW LOOPS	RT HEMICOLEC	-	ATTON 20 POD	
49.	PETER	33	M	89177	-	ABD PAIN 3 ½ DAYS	DIFF GUARD RIGID	SHT	FF ABD	-	-	-	-	MES. TB/ MIN ASCIT	MES NODE BX	-	ATTON 14 POD	
50.	MOHAN	21	M	60221	+/ 1 MONT	ABD PAIN/ABD DIST 2 ½ DAYS	ABD DIST/ DBS	-	DIL SMA BOW	DIL SMA BOW				ILE STIRC	RESEC ANAS	-	ATTON 15 POD	

51.	PRIYA	17	F	53267	-	ABD PAIN/ABD DIST 1 ½ DAYS	ABD DIST/ DBS	-	DIL SMA BOW	DIL SMA BOW				ILE STIRC	RESEC ANAS	-	ATTON 13 POD	
52.	SUNDARAM	26	M	78156	+/1 MON	FEVER/ABD PAIN 1 ½ DAYS	GUARD/ RIGID		N	-	-	-	-	MES. TB	MES. NODE/ OMEN BIOP	-	ATTON 21 POD	
53.	RAGAVI	26	F	57122	-	FEVER/ABD PAIN 2 ½ DAYS	DIFF GUARD RIGID	-	PROB TEND	-	-	-	-	MES. TB	MES NODE BX	-	ATTON 20 POD	
54.	ABDULLAH	28	M	49012	-	FEVER/ABD PAIN 4 DAYS	DIFF GUARD RIGID	-	PROB TEND	ASCIT FOR EVAL	-	-	-	MES. TB/ MIN ASCIT	MES. NODE/ OMEN BIOP	-	ATTON 14 POD	
55.	KARUPANNAN	27	M	73671	+/3 MON	ABD PAIN/ABD DIST 2 DAYS	ABD DIST/ DBS	-	DIL SMA BOW	DIL SMA BOW	-	-	-	ILE STIRC	RESEC ANAS	ANAS. LEAK/SEPT/DEATH		
56.	MARY	31	F	11903	-	ABD PAIN/ABD DIST 3 ½ DAYS	ABD DIST/ DBS	-	DIL SMA BOW	-	-	-	-	ILE STIRC	RESEC ANAS	WOUND INFECTION	ATTON 15 POD	+
57.	VELMURUGAN	42	M	29011	-	ABD PAIN/ABD DIST 5 ½ DAYS	ABD DIST/ DBS	-	DIL BOW LOOPS	? GROWTH TRANSV COLON	-	-	-	TRAN COLON MASS	EXT RT HEMICOLEC/ PROX. DIV	WOUND GAPING	ATTON 27 POD	
58.	RAGHU	28	M	51098	-	FEVER/ABD PAIN 3 ½ DAYS	GUARD/ RIGID	-	N	-	-	-	-	MES. TB	MES. NODE/ OMEN BIOP	-	ATTON 14 POD	
59.	THOMAS	28	M	30991	+/1½ MON	ABD PAIN/ABD DIST 3 DAYS	ABD DIST/ DBS		DIL SMA BOW					ILE STIRC	RESEC ANAS	WOUND INFECTION	ATTON 15 POD	
60.	PERUMAL	47	M	40914	-	FEVER/ABD PAIN/ MASS 10 DAYS	MASS RIF	-	APPEND ABSCESS/? MASS	? PSOAS ABSCESS/ ? IC MASS	-	-	-	RETRO PERIT NODES IN RIF	NODE BX	WOUND INFECTION/RTI	ATTON 16 POD	+

61.	DHAMODHARAN	47	M	10319	-	FEVER/ABD PAIN 3 ½ DAYS	GUARD RIGID	-	F.F	ASCIT FOR EVAL	-	-	-	PERIT ABSCESS	PUS C/S MES. NODE/ OMEN BIOP	WOUND INFECTION	ATTON 17 POD	
62.	RANI	32	F	81023	+/ 3 MON	ABD PAIN/ABD DIST 4 ½ DAYS	RIF MASS /ABD DIST/ IBS	DMSHT	RIF MASS FOR EVAL	-	-	-	YES	IC TB/DIL SMA BOW LOOPS	RT HEMICOLEC/ DIV ILEO	-	ATTON 17 POD	+

ANNEXURE C:

QUESTIONNAIRE

PATIENT DETAILS:

ON ADMISSION:

Name :

Age :

Sex :

IP No. :

Duration of symptoms :

Co-morbid illness :

DM : yes / no IHD / CAD : yes / no

HT : yes / no

H/O previous abdominal pain in recent past: yes/no if yes, duration:

H/O taking drugs for TB : yes / no

Whether the patient completed the duration of treatment: yes/no

If completed treatment, time since the completion of treatment:

CLINICAL EXAMINATION:

Pulse :

BP :

Anemia : yes / no

Icterus : yes / no

Pedal edema : yes / no

CVS: P/R:

RS:

P/A:

INVESTIGATIONS :

Hemogram:

Renal Function Test:

Liver Function Test:

HIV 1&2:

USG ABD:

CECT ABD:

INTERVENTION DONE: -

DETAILS OF INTERVENTION:

POST OP : -

GROSS MORPHOLOGICAL APPEARANCE OF THE SPECIMEN:

HISTOPATHOLOGICAL SPECIMEN REPORT